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LXIV

INFLAMMATORY STRICTURE OF THE ESOPHAGUS APPARENTLY PRESENT AT BIRTH

REPORT OF A CASE

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Congenital abnormalities of the esophagus are encountered fairly frequently, especially in white female infants. The usual developmental defect consists of communication of the lower end of the esophagus with the air passages, at or near the bifurcation of the trachea, with the upper segment terminating as a blind pouch. In recent years early recognition of this deformity and prompt operative treatment with anastomosis of the two segments of the esophagus has resulted in the salvaging of many children. Not infrequently narrowing occurs at the site of anastomosis with resulting difficulty in swallowing; but this is readily relieved by passing dilating sounds, over a previously swallowed silk thread, through the area of contraction. The passage of a 30 F. sound usually suffices to provide complete and permanent relief from dysphagia.

The second most frequent congenital defect is partial obstruction of the lower portion of the esophagus in the vicinity of the bifurcation of the trachea by a diaphragm that consists of reduplication of

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the mucus membrane of the tube. The aperture through this diaphragm is usually quite small and frequent regurgitation of feedings dating from birth usually leads to the detection of obstruction in the esophagus at an early age. When the opening is somewhat larger dysphagia is not apparent until the child begins to ingest semisolid or solid food. In other instances the presence of a narrowed area in the esophagus may not be identified until much later in life.

In older patients a stricture that may be the result of congenital narrowing is usually thought to be acquired and of inflammatory origin. As a matter of fact inflammatory reaction at the site of congenital narrowing of the esophagus may explain the occurrence of progression of dysphagia in early or middle adult life. Therefore, differentiation of congenital from acquired stricture may at times be difficult or impossible.

Partial obstruction of the esophagus that produces symptoms at or soon after birth is almost always the result of congenital stricture. The area of constriction is short and the passage of a 30 F. sound through the stricture results in complete and permanent relief from dysphagia.

The following case is recorded because the stricture in the esophagus that was thought to be congenital proved to be inflammatory in origin.

REPORT OF A CASE

The patient, a white baby girl three and one-half months of age, weighed 6½ pounds at birth and at the time of examination the weight had increased to 12 pounds. She had had spells of respiratory difficulty when nursing that began at birth and were associated with cyanosis. Bottle feedings were more readily tolerated. Cereals were regurgitated immediately after being ingested and there seemed to be excessive mucus in the throat. Because of apparent dysphagia roentgenoscopic examination was made of the esophagus and a stricture was revealed (Fig. 1). This obstruction was thought to be of congenital origin.

No difficulty was encountered in having the baby swallow a thread and on April 21, 1954, a stricture located 5½ inches from the upper gum margin was easily dilated with a sound sized 29 F. without bleeding. Immediately following dilation the child was able to swallow normally and evidence of excessive secretion in the throat disappeared.

For two weeks deglutition was normal and the patient gained two pounds in weight, but then swallowing became more difficult

and a second dilatation was done on May 19, 1954. At this dilation a 35 F. sound was passed through the area of obstruction with a little more resistance than at the first treatment and without bleeding. After dilatation the child took a feeding normally and then slept for almost an hour. On awakening she was evidently in pain and the temperature was elevated. Although there was no evidence of subcutaneous emphysema or air in the pleural spaces or mediastinum, the ingestion of iodized oil revealed a small sacculation at the site of the stricture (Fig. 2). This was interpreted as a rupture and operation revealed a perforation of the esophageal wall one-half inch in length.

The split in the esophagus was thought to be closed satisfactorily, but as the operation was being concluded the heart stopped beating. Normal rhythm was re-established by intermittent compression. Soon thereafter twitching was noted of the muscles of the face and upper extremity. Cheyne-Stokes respiration then developed with prolonged periods of apnoea. Death occurred eight hours after operation.

Post mortem examination revealed an irregular inflammatory stricture of the lower portion of the esophagus approximately 1 inch long with perforation and localized mediastinal infection (Fig. 3).

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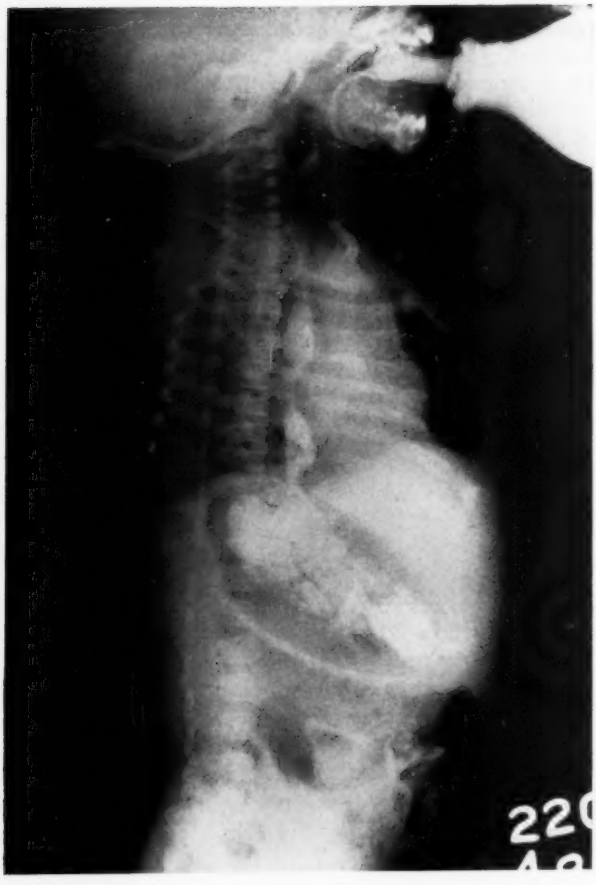

Fig. 1.—Showing stricture in the lower portion of the esophagus.

Fig. 2.—Sacculation at site of stricture.

Fig. 3.—Annular inflammatory stricture with rupture.

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TERATOID TUMOR OF THE NASOPHARYNX IN A NEW BORN

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This case history report of a teratoid tumor in the nasopharynx of a newborn is of interest for two reasons: 1) a rather rare tumor occurring in a rather unusual location; 2) the extreme youth of this particular patient.

This full term female baby was born on March 26, 1954. The birth weight was eight pounds, four ounces. At the time of birth no abnormalities were noted. The child was handled in the routine manner and transferred to the newborn nursery. On admission to the nursery, the nurse noted that the child had an unusual amount of mucus in the nose and throat. The initial feeding offered caused the baby to choke and become cyanotic. The baby was unable to swallow formula or fluid.

The physical examination was normal except for the following findings: The oral pharynx revealed a small white polypoid mass which protruded about 3 mm from underneath the left posterior portion of the soft palate. When the tongue was depressed and this mass was grasped with a small hemostat it was found to extend into the nasopharynx. It was attached by a pedicle to the region of the left eustachian tube. The tumor had smooth, pale white surfaces. It was firm and lobulated. When the tongue blade was removed from the area of the posterior portion of the tongue and the tension released, the mass slid back into the nasopharyngeal area. Likewise, all attempts to give the child water caused the mass to slide back down and obstruct the airway; and the child would gag and become cyanotic as the result of attempts to swallow.

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Since the tumor offered itself as a mechanical obstruction to deglutition, further investigative studies of the nasopharynx and esophagus could be detrimetal to the infant. It was obvious that the obstruction would have to be removed surgically if the baby was to eat.

An anesthesiologist was available. However, by immobilizing the baby with a sheet, a nurse keeping the head fixed with her hands, no anesthesia was necessary. In surgery the child was placed in the tonsil or semi-Rose position and the mass was grasped with a hemostat and retracted down into the pharyngeal area. This tension demonstrated an elongated pedicle which led up into the region of the fossa of Rosenmuller on the left side. Before proceeding with the surgical removal, attempts to aspirate the tumor showed it to be cellular with no cystic characteristics. A 2-0 chromic ligature was applied at the base of the pedicle, and with a Tyding's snare loop distal to the ligature, the tumor mass was removed. There was very little bleeding. The tissue was sent to the laboratory for histological examination and diagnosis.

Following removal of the tumor a small polyethylene tube was passed into the stomach easily. This was done to establish the patency and continuity of the esophagus and rule out other possible congenital anomalies of this structure.

The baby was returned to the nursery and enjoyed regular formula every three hours. There were no swallowing difficulties nor was there any apparent swelling or respiratory distress. At the time of discharge on the fourth post-operative day, she had almost regained her birth weight. At the present time, the child is doing satisfactorily and is gaining weight. The last examination at the age of four and one-half months showed no evidence of tumor.

Pathological report by P. M. Smith, M.D. of the Children's Hospital Staff:

"*Gross:* The material consisted of two gray-white tumor masses from the nasopharynx. The larger piece is 1.6 by 1.2 by 1.0 cms. Most of the surface is smoothly rounded. At one end a dissected surface of 0.7 cms in diameter is seen. The second piece is 1.2 by 1.1 by 0.5 cms. A broad end is slightly discolored blue-gray but most of one surface is covered by a smooth gray-white membrane. The opposite surface is dissected and is gray and granular appearing.

Microscopic: Both pieces are covered by a thin well differentiated stratified squamous epithelium of the dermal type. The stratum corneum is relatively wide and there is a thin stratum granulosum. Numerous skin appendages are present including sebaceous

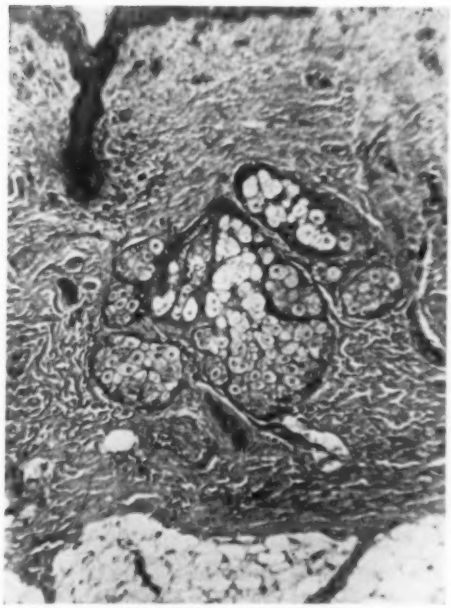

Fig. 1.—Photomicrograph of teratoid tumor removed from nasopharynx. Note the squamous epithelium, fat tissue, glands and collagen connective tissue. (x 100)

glands, hair follicles and sweat glands. The central portion of the masses is composed largely of lobules of fat separated by strands of collagenous fibrous tissue which include skeletal muscle fibers. Toward the base the skeletal muscle fibers are more numerous and in this region there is a small island of cartilage and a group of mucous glands." (Fig. 1)

Nasopharyngeal tumors in the newborn are rare, and teratoid tumors in this region are even more rare. Foster,[1] in 1944, reported a similar tumor in a child 19 days old. The symptomatology and objective findings in his patient were very similar to the findings noted in our case. As far as we know, this 18-hour-old baby is the youngest patient with this type of tumor reported in the literature.

450 Sutter St.

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1. Foster, J. H.: Congenital Dermoid Tumor of the Nasopharynx, Annals of Otology, Rhinology and Laryngology 53: 578-80 (Sept.) 1944.

LXVI

NASOPHARYNGEAL CARCINOMA

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The strikingly high incidence of nasopharyngeal cancer in Chinese as reported by Martin and Quan,[1] has prompted us to undertake the present study to find out if it has any racial predilection for all orientals or if there are any special factors operating in China to account for this heavy incidence, i.e. 18 per cent of all cancer cases.[2]

This present inquiry extends over a period of three years and takes cognizance of all cancer cases examined in the Victoria Jubilee Hospital, attached to the Medical College, Amritsar. The proportion of nasopharyngeal cancer to all cancer cases in this hospital was 1.8 per cent for outpatients (16 cases out of 868) and 2.3 per cent for inpatients (16 cases out of 688). These figures correspond fairly well with the figures of American hospitals which report about 2.5 per cent of all cancer cases (McPhee and LaCroix[3]). Even the latest figures of the Tata Memorial Hospital, Bombay, which attracts cancer cases from all over India put the average incidence of nasopharyngeal cancer at 1.7 per cent.[4] The figures of this hospital speaking for the Punjab and of the Tata Memorial Hospital representing the whole of India are a clear proof against any racial predilection of this disease for all orientals.

There are, however, certain very peculiar features of this disease which prevented its early diagnosis in the recent past. Even in the West with all the modern facilities for early diagnosis, only 10 years ago the incidence of the disease was worked out at 0.3-0.4 per cent (Godtfredsen[5]). The Lahey Clinic in America has reported only

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26 cases during the last 10 years. The outstanding features evading early diagnosis are:

(1) The inconspicuous and asymptomatic nature of the growth in the early stages. The original tumor is just a small nodule, silent and flat, covered with a healthy and unbroken mucosa or submerged in the overgrowth of lymphoid tissue normally present in excess in the nasopharynx. This tumor is easily missed on routine posterior rhinoscopy and if you suspected one and performed the biopsy, the first report may not give any clue of the original disease due to overgrowth of lymphoid tissue. This is in strong contrast with nasopharyngeal fibroma which is voluminous and exophytic and fills up not only the nasopharynx but also encroaches upon all the neighboring structures i.e. depressing the soft palate, obstructing choanae and blocking the eustachian tubes. The only sure way to make an early diagnosis is not to miss the digital examination of the nasopharynx on the slightest suspicion of symptoms and signs referred to eyes, ears, nose throat, neck and brain.

(2) The fact that the original tumor remains small and quiet but the metastases into the lymph glands are early and extensive. The upper deep cervical glands of the posterior group are affected quite early and 60 per cent of the patients complain of these glands and nothing else. These glands are so different from secondary cancers of other organs that one is apt to miss their true nature. They are firm but not hard, large and freely mobile with the skin normal in colour and easily displaceable over them. They are discrete in the earlier stages with very little periadenitis and grow rapidly. They lie deep under the upper end of the sternomastoid or under cover of the middle of the sternomastoid and produce stiffness of the neck and difficulty in opening the mouth. Bilateral involvement of the cervical glands is common in nasopharyngeal cancer which is otherwise rare in unilateral cancerous disease. These glands, therefore, resemble more the inflammatory ones and are often diagnosed as tuberculous or as Hodgkin's disease, as there is also sometimes some elevation of temperature with them. On the other hand this is rather a late sign as the usual expectation of life after the appearance of glands is 12-13 months.

(3) The quiet and unobtrusive growth and spread in directions which one hardly suspects, thus missing the disease. It has special predilection for the orbit and approaches it extracranially through the infratemporal fossa and the inferior orbital fissure or intracranially through the foramen lacerum and along the cartoid groove to the superior orbital fissure. The latter route is common

as the usual site of the origin of the growth is the fossa of Rosenmüller which is situated just underneath the foramen lacerum. It eats through the basi-occiput and involves the jugular foramen and gives a combination of symptoms and signs grouped under various syndromes of the jugular fossa. It may also eat through the basisphenoid and involve the sphenoidal sinuses. It rarely extends forwards and blocks the choanae, unlike the nasopharyngeal fibroma which almost always blocks them. This shows how cleverly it conceals its own original identity and misleads the unwary unless he keeps nasopharyngeal carcinoma at the back of his mind and makes a routine digital examination of the nasopharynx.

(4) Difficulties of proper examination. As 60 per cent of these patients come with enlarged glands in the neck, unilateral or bilateral, resulting in trismus, the mirror examination of nasopharynx is very difficult and even when an examination is made, the appearances are not at all suggestive of malignancy as it is very late in its course that it ulcerates and fungates.

(5) Biopsy is difficult and not always conclusive at first attempt. It is only when the ulcerated growth is present that an early diagnosis is possibly by biopsy.

THE PRESENT STUDY

The present study comprises 16 cases; some of them of special interest will be reported in detail while all are discussed in brief.

The total number of malignant tumors of all varieties seen at the outpatient department of the V. J. Hospital during the year 1949 to 1951 was 868 out of which 16 were nasopharyngeal; a percentage of 1.85. Out of these 688 sought admission as inpatients; the percentage is 2.3. In the same period we also treated 16 cases of malignant tumors of the tonsils. Out of them 10 were lympho-epithelioma, and 6 were squamous cell carcinoma. Thus its incidence in this country runs pari passu with that of tonsillar malignancy. The incidence of nasopharyngeal carcinoma in relation to the malignant tumors of other parts of the body as shown by the statistics of this hospital is given in Table 1. This shows that it is much lower than in China. The explanation offered for the heavy incidence in Chinese is the peculiar habit of smoking of opium by the Chinese and exhaling the fumes through the nose, although this seems unlikely since opium contains no carcinogenic substance. The only effect may be chronic irritation. This irritation should be more in sulpha (charas or crude resin of cannabis) and opium smoking sadhus in India in whom the incidence of nasopharyngeal carcinoma

TABLE 1.
PERCENTAGE OF NASOPHARYNGEAL CARCINOMA IN COMPARISON WITH OTHER MALIGNANT TUMORS IN VICTORIA JUBILEE HOSPITAL, AMRITSAR.

YEAR	TOTAL MALIGNANT GROWTHS	MALIGNANT TUMORS OF CERVIX AND UTERUS	MALIGNANT TUMORS OF EAR, NOSE AND THROAT	MALIGNANT GROWTHS OF TONSILS	MALIGNANT GROWTHS OF LARYNX	MALIGNANT GROWTHS OF NASOPHARYNX
1949	182	120	36	5	2	4 (2.2%)
1950	273	177	24	6	2	6 (2.2%)
1951	233	131	38	5	4	6 (2.57%)
Total	688	428 (62.2%)	98 (14.2%)	16 (2.3%)	8 (1.15%)	16 (2.3%)

is in no way higher than in the general public. However, the higher incidence of nasopharyngeal carcinoma in Chinese living in the United States suggests that the tendency is racially inherited rather than due to environment.[1]

Out of 16, 13 were males and 3 females, giving a preponderance in the males with 81 per cent. This corresponds closely with the findings of Martin[6] (1948) who also found it more frequently in males (81%) than females. Ewing[7] cites an incidence of 80% in males.

Seventy-five per cent of the patients were below 40 years of age and the maximum incidence was between 31 to 40 years (50%) with the youngest patient seen at the age of 7 years and the oldest, 70 years. From this it is clear that another peculiarity of this tumor is that it does not spare even the very young. These figures closely resemble the Chinese statistics as reported by Digby[4] from Hong Kong in which 50 per cent were seen between 31 to 45 years (Tabe 2). In Western countries maximum incidence is between 41 and 50. This discrepancy may easily be explained by the lower expectation of life in Indians.

Out of 16, three hailed from the cities with an average population of 1,200,000. Thirteen cases came from the rural areas with the population of 4,800,000. It is, therefore, clear that urbanization has no effect on the incidence of this disease, the percentage for the total population being the same in both urban and rural areas.

TABLE 2.
MALIGNANT GROWTH OF NASOPHARYNX IN DIFFERENT AGE GROUPS IN COMPARISON TO OTHER NATIONS.

AGES	NO. OF CASES	PERCENTAGE IN OUR CASES	PERCENTAGE IN CHINESE	PERCENTAGE IN AMERICANS
1 to 10	1	6.25%		
11 to 20	2	12.5%	7.7%	Under 20 - 5.3%
21 to 30	1	6.25%	15.5%	Under 30 - 9%
31 to 40	8	50.0%	36.9%	Under 40 - 21%
41 to 50	2	12.5%	27.09%	
51 to 60	1	6.25%	12.3%	
61 to 70	1	6.25%	2.4%	
Minimum age:		7 years		16 years
Maximum age:		70 years		76 years

SYMPTOMATOLOGY

As already stated the varied symptoms produce a picture which suggests anything but a disease of the nasopharynx and, therefore, unless you keep this attic of the nose at the back of your mind in arriving at a diagnosis you will invariably be led astray. The various symptoms mentioned by our patients are listed below:

Sixty per cent peresented themselves with glandular mass or masses in the neck, whereas they were present in 90 per cent on examination—70 per cent bilateral and 20 per cent unilateral. Sixty per cent of the patients had unilateral headache which varied in severity. The headache was referred to the temporal and frontal areas (first and second division of the 5th cranial nerve). It may be on the vertex and on the occipital regions also. Two patients had unilateral earache which was variable in intensity. Two complained of progressive and unilateral deafness. It was of middle ear type and was associated with chronic suppurative otitis media in one case. In another mastoidectomy had been done before for the referred ear symptoms. Eight patients had blood stained sputum which was never a first symptom. Six complained of epistaxis which was the first symptom in only one case. Nasal obstruction was the

first symptom in only one case whereas four other patients also had this trouble. One patient developed exophthalmos and in two others there was involvement of the third nerve i.e. ptosis and diplopia. Hemiparesis with fits of unconsciousness due to intracranial extension of the growth was present in one case. One patient had dysphagia as the first symptom though mild disturbance in swallowing due to involvement of jugular foramen group of nerves was present in three cases. One had nasal twang in his voice due to mechanical causes while clear cut hoarseness was present in one case. In two cases there was paresis of the soft palate. Five patients had lost weight while general weakness gradually came on in all, late in the course of the disease. Less common complaints were regurgitation through the nose (1 case), stiffness of the neck (1 case) foul breath (1 case), and sore throat (1 case). In one case lumps in the axilla and abdomen were late symptoms. In this very patient fluid in the pleural cavity was a terminal symptom. Lump on the sternoclavicular joint was present in one patient who developed paresis of the legs subsequently.

This varied symptomatology needs the close collaboration of the rhinologist, the dentist, the ophthalmologist, the neurologist, the radiologist, the pathologist and the physician for a proper diagnosis. No wonder the right diagnosis was missed in such large numbers in all countries, only 20 years ago. It may not be out of place to mention one case of lympho-epithelioma of the nasopharynx, reported by Thompson,[8] diagnosed on the fourth admission, and after a period of ten months following the first examination due to symptoms referable to other organs. How could one dream of the nasopharynx as the cause of hoarseness and even of exophthalmos.

CLINICAL SIGNS

Peculiar features have already been mentioned. Thorough examination of ear, nose and throat must be carried out with special emphasis on digital examination. Examination of throat may show no abnormality of the pharynx or there may be a bulging of the posterior wall on one or both sides, corresponding to the outer lumps. Blood may be trickling from the nasopharynx. One tonsil may be pushed medially. Anterior and posterior pillars, uvula, soft palate and tongue may be normal, but when the growth arises from the dorsum of the soft palate it may be depressed or in rare cases when the growth is exophytic, it may depress the corresponding soft palate. The various cranial nerves coming out of the jugular and condylar fossae may be involved resulting in paralysis of the soft palate and the vocal cords. The patient may complain of pain in the ears but

on otological examination nothing abnormal may be detected. The pain in the ear is a referred one and deafness is of the conductive type.

LABORATORY INVESTIGATIONS

Total and differential leucocyte count, erythrocyte sedimentation rate, Kahn test and Mantoux test will help to distinguish the glandular swelling from Hodgkin's disease, tubercular, syphilitic and infective lymphadenopathies. X-ray of the nasopharynx—lateral, axial and half axial should be taken. Destruction is the main feature and if one finds unilateral destruction of the apex of the petrous bone or basiocciput or widening of the foramen lacerum or involvement of jugular foramen, the suspicion is confirmed. X-ray of the chest should be made to exclude tuberculosis or secondary lues. Biopsy should be made from the deeply removed specimen. If one is negative it should be repeated.

TREATMENT

The treatment of choice is irradiation. The usual method is deep x-ray therapy. Some combine it with radium needles or seeds. The practice in our hospital is 2500 to 3000, r, in air in 15 days with 10 sittings, administered from a portal of 20 cm with a skin target distance of 25 cm and a filter of Cu 0.5 and aluminum 0.5 mm with 250 kv and 10 M.A. to be repeated after 3 months. The results have not been satisfactory, perhaps a narrower portal with larger dose may be more successful but it is the early diagnosis which matters most, for early irradiation will give us a greater percentage of five year cures. It is too early to speak of five year cures in our cases as the figures are for three years only (five patients have died so far).

REPORT OF CASES

Although this series consists of 16 cases, detailed histories of only interesting ones are presented.

CASE 1.—B. S., a male aged 42, peasant, was admitted to this hospital on August 28, 1951. His complaints were defective hearing which started on the right side, pain in the temporal region and vertex—all in two months; swelling on both sides of the neck for the past 25 days; epitaxis and deterioration of general health—duration 20 days.

Examination of the neck revealed a lump under the middle of the right sternomastoid muscle about 3 inches by 2 inches in size. There was a tendency towards hardness in the upper part. The mass

was freely movable over the structures underneath and the overlying skin was healthy and mobile. There was no softening or tenderness. A similar but smaller mass was present on the opposite side of the neck in the same location. Examination of the throat showed left sided paresis of the soft palate and blood-stained discharge on the posterior wall of the nasopharynx. Digital examination of the nasopharynx revealed the presence of an ulcerated growth. The tumor was somewhat hard and arose from the middle of the roof of the nasopharynx and extended laterally on both sides. The growth started bleeding after examination. Bleeding was never profuse. There was marked middle ear deafness on both sides and the drums were slightly retracted.

Radiography showed a big soft tissue mass in the nasopharynx, in the lateral view. No bone destruction could be seen in the axial view. Histopathological examination revealed lympho-epitheloma. Deep x-ray exposures were given as already described. The neck glands disappeared and the nasopharyngeal mass became very small. There was relief of headache and no further bleeding from the nose during three months' stay in the hospital. The patient was discharged on December 3, 1951, and returned on February 12, 1952, complaining of tenderness, pain and tenseness on the right side of the face, difficulty in swallowing and mastication, swelling on the right side of the neck, blurring of vision, diplopia and headache.

On examination discrete hard glands could be palpated under the middle of the sternomastoid and in the subclavian triangle on both sides. There was internal paralytic squint in the right eye without involvement of any of the other cranial nerves. Digital examination of the nasopharynx revealed a nodule in the middle line of the roof. The nodule was smaller in size than before. It was removed and sent for histopathological examination. Pathologist again reported lympho-epithelioma. Axial view of the skull did not show any bony erosion. The patient was put on deep x-ray therapy.

On March 4, 1952, he complained of greater difficulty in mastication and swallowing. There was ptosis in the right eye and paresis of the external rectus muscle developed in the left eye also (Fig. 1). Pain over the right side of the face was more severe. On March 11, there was complete anesthesia over the distribution of the 5th cranial nerve of the right side but the patient complained of severe pain over that area. The right eye ball was fixed and the pupil was dilated. It did not react to light and ptosis had increased. On March 14, 1952, there was marked proptosis of the right eyeball which was fixed. As the disease was showing a rapid progress intracranially

Fig. 1.—Bilateral convergent squint due to involvement of abducent nerves.

towards the superior orbital fissure, the patient became weak and the general condition alarming, deep x-ray therapy was discontinued after consultation with the radiotherapist. The patient left the hospital on April 7, 1952, in the same condition as on March 14, 1952 and died a week later.

Case 2.—N. S., a male aged 70, farmer, reported to the hospital on October 21, 1950, complaining of pain in the left temporal region, blood stained sputum, and pain in the left ear, all for the last six months; swelling of the left half of the face and general weakness—duration 4 months.

On examination the neck showed hard, tender and discrete glands on the upper part of the left side of the neck. There was one enlarged gland on the right side also. These glands did not show any softening or breaking. Digital examination of the nasopharynx revealed a small friable growth on the left side. The mucous membrane over it was ulcerated and the growth was confined to the left side.

Roentgenograms of the skull showed rarefaction of the petrous part of the left temporal bone. The pathologist reported lymphoepithelioma of the Schminke type. The patient was put on deep

Fig. 2.—Involvement of the left oculomotor nerve leading on to ptosis of the left eye.

x-ray therapy but after ten exposures the x-ray plant went out of action and the patient was asked to report again after a month or so. With this incomplete treatment his symptoms vanished. On April 10, 1951, he reported to the hospital for completion of the course of deep x-ray therapy. At that time he had no complaints and examination of the nasopharynx did not reveal any growth but remaining exposures of deep x-ray therapy were given. On August 27, 1951, he returned to the hospital with paralysis of the 3rd cranial nerve on the left side (Fig. 2) and paralysis of the left side of the palate showing intracranial extension of the growth. He was given another course of deep x-ray therapy. The patient died at home six months later.

CASE 3.—R. D., a boy aged 12 years, developed stiffness of the neck after lifting a heavy weight—8 months previously. For this he got treatment from some general medical practioner but without any relief. His other complaints were, headache since seven and

Fig. 3.—Post-operative paralysis of the left facial nerve and swelling on the left side of the neck and face.

one-half months, difficulty in opening the mouth since seven months, pain in the left ear and rapid loss of weight since 6 and three-fourths months. Because of ear pain he was given penicillin injections by some general medical practitioner but as they proved ineffective he was put on penicillin and streptomycin. Skiagram of the mastoids revealed haziness of the left mastoid cells. As the medicines did not improve his condition, mastoidectomy was done, which was followed by facial palsy (Fig. 3). During this period a gland was noticed in the middle of the neck under cover of the left sternomastoid. The gland was very tender and painful.

The patient was first examined in the outpatient department of the hospital on April 11, 1951. There was a solitary, enlarged and tender gland under the middle of the left sternomastoid. It was freely mobile and the skin over it was healthy and movable. There were no signs of softening and calcification. Examination of the left ear revealed a large central perforation with slight mucopurulent discharge. Patient had marked trismus. Digital examination of the nasopharynx showed a small ulcerated growth in the left half of the nasopharynx. It bled readily on touch and was friable. Cancer was diagnosed and the patient was admitted to hospital on May 2, 1951. Roentgenogram of the base of the skull in

Fig. 4.—Rarefaction of the petrous part of the right temporal bone seen in the axial view of the skull.

the axial position showed erosion of the bones in the neighborhood of the nasopharyngeal end of the eustachian tubes. Histopathological report was lympho-epithelioma of the transitional-cell type. He was put on the usual course of deep x-ray therapy to which he responded very favorably and was free from all the symptoms after that.

On August 22, 1951, he again reported to this hospital for routine check up. Digital examination of the nasopharynx did not show any evidence of recurrence but a cervical gland on the left side was still tender.

In October 1951 the growth had recurred and he proceeded to Bombay for treatment where he expired before the course of deep x-ray therapy could be completed.

CASE 4.—D. S., a male aged 36, farmer, was admitted to this hospital on June 19, 1951, complaining of epistaxis for one and one-half years, partial obstruction in breathing since 14 months, headache and lump on the right side of the neck for one year.

Fig. 5.—Markedly cachetic patient with a big lump on the left side of the neck (photograph taken one day before death).

The cervical glands in the upper part of the right side were enlarged. They were discrete, one-half inch in diameter, firm and freely movable over the underlying structures. The skin over them was healthy and mobile. There was softening or breaking down. On digital examination of the nasopharynx a small, friable growth was palpated on the right side. It bled on examination. Roentgenogram of the skull (axial view) showed rarefaction of the petrous part of the right temporal bone (Fig. 4). A biopsy specimen was diagnosed as epidermoid carcinoma. The patient was given a complete course of deep x-ray therapy which relieved him of all the symptoms.

He again reported to the hospital on February 21, 1952. On examination of the neck there were enlarged glands under the upper part of the sternomastoid. Those on the right side were larger in size. Examination of the nasopharynx with posterior rhinoscopy mirror revealed a big tumor filling the upper part of the pharynx. The growth was greyish pink and had an irregular surface. The

eustachian tubes were blocked and the choanae could not be visualized. The patient was given a second course of deep x-ray therapy. He died at his home one month later.

CASE 5.—G. K., a girl aged 12 years, reported to the outpatient department on July 19, 1950, complaining of excessive salivation for one and one-half months, inability to speak properly and a nasal twang for the past month, difficulty in swallowing for the past 12 days, regurgitation through the nose, pain in the throat and ears, a mass in the mouth and low grade pyrexia for the preceding 12 days.

A hard and fixed gland, 1 cm x 1 cm in size, was palpable at the left angle of the lower jaw. It could be moved over the underlying structures and the skin over it was healthy and mobile. The gland was tender and there were no signs of softening. Examination of the throat revealed a large, pinkish, lobulated, firm, friable and fungating growth arising from the soft palate and the uvula. There was not much local induration and infiltration. The growth was large enough to obstruct breathing and the patient kept the mouth constantly open. The tonsils and anterior pillars were free from the growth. She was admitted as a case of epithelioma of the dorsum of the soft palate.

A roentgenogram of the skull in the lateral position revealed a growth arising from the posterior part of the palate and hanging down into the oro- and hypopharynx, almost completely obstructing the cavity. Biopsy produced a report of lymph-epithelioma of the Regaud type. The growth was excised under general anesthesia and she made an uneventful recovery. She was relieved of all the symptoms and was dischargd on August 5, 1950. She was advised to report for deep x-ray therapy but she did not turn up.

Three months later she returned to the hospital with the same complaints and an almost similar growth which was smaller in size. Growth was again excised and a full course of deep x-ray therapy was given with which her complaints disappeared.

One year later she again presented herself in markedly cachectic condition, having a big lump on the left side of the neck (Fig. 5). Examination of the nasopharynx was not possible as she could not open the mouth at all. She was admitted to the hospital where she expired on the next day.

COMMENT

The incidence of nasopharyngeal carcinoma in the Punjab is 1.85 per cent and this corresponds with the all-India figures reported by the Tata Memorial Hospital, Bombay. This definitely sets at

rest the view that the occurrence of nasopharyngeal carcinoma is very high in all Orientals. The heavy incidence in Chinese will have to be accounted for in some other way This cancer has an earlier age incidence than carinoma of other parts of the body. It is not rare in children. So far as the sex incidence is concerned, males preponderate (81 per cent).

Most of the patients came late. Sixty per cent of them presented themselves because of enlargement of the cervical glands although on examination these were present in 90 per cent of the cases. The glands were entirely different from the metastases of other malignant tumors and, therefore, were mistaken for tuberculosis or Hodgkin's disease. It is emphasized that in all cases of cervical lymphadenopathy, unilateral, bilateral, especially of the upper deep cervical (posterior group), the nasopharynx must be examined by digital examination, as the primary growth is very inconspicuous.

Various combinations of symptoms referable to eyes, ears, nose, throat, brain, bones of the skull and cranial nerves for example hoarseness, regurgitation from the nose, exophthalmos, trigeminal neuralgia and jugular foramen syndromes, may all have their origin in the nasopharynx, and call for digital examination. Because of this bizzare symptomatology and of the growth being so unobtrusive whereas its metastases to the neighboring structures are so prominent, the wrong diagnosis is very frequent. The local extensions are to the bones of the skull and through the various foramina to the interior of the skull.

The difficulty of diagnosis is again emphasized.

The triad of x-ray diagnosis—destruction of the petrous apex, basi-occiput and enlargement of foramen lacerum, was present in late cases.

Biopsy is not easy and sometimes will have to be repeated. The histopathology is rather confusing as there is always an admixture of epithelial and lymphoid cells. It is the varying proportion of these with necrosis of superficial layers and supervention of infection over the fungating mass that complicates the diagnosis. Different pathologists will give different views on the same specimen.

The prognosis is not hopeful as the patients usually come late because the growth is painless, small and situated in an area inaccessible to routine examination. The early metastasis and extension upwards to the brain make the prognosis grave and the inaccessibility of the area to radical surgery and the still undeveloped radiotherapy make it still graver. The various statistics after five years are rather

disappointing while in our series even three years cures were much too few. The effect on general health is rather late as even with large lumps in the neck the patients do not lose either their color, freshness or weight, because of slight absorption of toxins from the nasopharyngeal mucosa.

SUMMARY

1. A detailed study of sixteen cases of nasopharyngeal carcinoma seen in three years (1949-51) is presented emphasizing the close correspondence between the incidence of nasopharyngeal carcinoma in India and in the West.

2. The importance of digital examination of all patients is stressed.

Nasopharyngeal carcinoma must be considered as one of the causes of cervical lymph adenopathy due to chronic or specific inflammatory lesions. To miss the diagnosis is an unforgivable mistake.

4. The difficulty of histopathological diagnosis is emphasized.

5. The gravity of the prognosis is stressed.

6. The limitations of radiotherapy are described.

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LXVII

EQUILIBRATORY ILLUSIONS IN AVIATION

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At the present writing it is unnecessary to assert the value of navigational instruments in aircraft flight. It is, however, of considerable interest to explain, on a physiological basis, the various illusory states which are encountered in flying personnel. These illusions may make manipulation of the aircraft a matter of real hazard to the flyer so that a good understanding of them is prerequisite to his safety.

Balance is maintained by an interaction of three systems which are relatively unrelated anatomically but which are quite closely related physiologically. These three system are the ocular, the aural, and the proprioceptive. They comprise what is known as the equilibratory triad, which may be represented as follows:

The *ocular* system provides orientation by visually aligning the individual with certain structures. In walking he will be conscious that he is upright, as are the trees and buildings, and perpendicular to the horizon. In the air, he can orient himself at right angles to the horizon (so that he is sitting as if on the land or in proper relation to another airplane). This is relatively easy in the day-time, but becomes impossible at night, in a cloud, a storm, or other inclement weather when the ability to see long distances is missing.

The *vestibular* system provides two types of orientation. The static organ is the utricle, which is thought to orient the individual by means of the distribution of the pressure of the otoliths upon the terminations of the utricular division of the vestibular nerve. Thus, the utricle is always aware of a tilt of the head and is not affected by the rate at which that tilt is produced. There is an added stimulation by gravity in which the vertical canals supplement the utric-

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ular function. There is no known gravitational function in the horizontal canals.

The *kinetic* part of the vestibule is the composite effect of the semicircular canals. Motion of fluid through the semicircular canals stimulates the neuroepithelium located in the crista of the ampullary end of the canal and produces the sensation of rotation. As these canals all open into the utricle, it is probably impossible to cause fluid motion in one canal without producing some, though slight, disturbance in the others. The two vertical canals actually have a crus communis, so action of one invariably produces motion in the endolymphatic fluid of the other. If a person is rotated in a given direction, in the plane of one of the semicircular canals, that motion will produce maximal stimulation of the canal on one side and minimal stimulation of the canal on the other, because fluid motion in the horizontal canals toward the ampulla produces maximal stimulus and away from it minimal stimulus. In the case of the vertical semicircular canals, the opposite is true, in that ampullipetal stimulation is minimal and ampullifugal is maximal. Thus, the vertigo produced by rotation is the resultant of the maximal stimulation of the one labyrinth, modified slightly by minimal stimulation of the other. This explanation for the function of the semicircular canals assumes motion of the endolymphatic fluid in the membranous labyrinth and does not take into account the fact, which some workers have clearly demonstrated, that the effect of head rotation on the fluid in the canals is entirely centrifugal (Armstrong,[2] Poppen[6]). It also ignores the effect of the so-called vortices which are formed when rotation does not occur in the exact plane of the canal.

It is this sort of stimulation with which flyers are concerned, because in no maneuver does the motion stimulate one ear or one canal alone. In all aerobatics and other forms of rotation, both ears are stimulated simultaneously as the head is turned as a whole. Another important criterion for stimulation of the vestibular apparatus is that the acceleration of the labyrinth must be greater than two degrees per second, per second. This is one of the important characteristics of the semicircular canals which results in misleading sensations to the flyer.

Laws governing the action of the semicircular canals:

1. Stimulation of a semicircular canal produces nystagmus in the plane of that canal (Ewald, Arellano[1]).

2. Nystagmus due to rotation is always in a plane at right angles to the rotation, without regard to the position of the head (Flourens, Arellano[1]).

3. After-nystagmus, the quick phase, is always in the opposite direction to the rotation (thus the show phase corresponds to the direction of the flow of the endolymph) (Arellano,[1] Barany,[4] Winston[7]).

4. Following labyrinthine stimulation, past-pointing and falling are in the direction of the flow of endolymph (the direction of rotation) while vertigo and nystagmus are opposite (Arellano,[1] Winston[7]).

5. In the horizontal canal of man, endolymph flow toward the ampulla is approximately twice as effective a stimulus as flow away from the ampulla. In the vertical canals, endolymph flow away from the ampulla is approximately twice as effective a stimulus as flow toward the ampulla (Ewald, Crum-Brown,[8] McNally[3]).

6. Rotation producing minimal stimulation of the semicircular canals must be at a rate of at least two degrees per second, per second (Dodge[9]).

The *proprioceptive* system orients the flyer with the location of his body in space. The nerve endings in the joints and the pressure sensitive areas of the skin, when properly stimulated, advise the flyer of the manner in which his extremities are arranged in space with relation to the other members of his body. This orientation has nothing to do with orientation in space relative to a landing field or any other object at a distance.

The forces which affect the three equilibratory systems mentioned above are numerous. There are probably few situations aloft in which the effects of one set of forces are noted entirely by themselves. There are, however, common situations in which the effects of one set of forces are more important than the effects of other forces. Physical forces which are important in the stimulation of the labyrinth are as follows:

Acceleration is one of the most important forces encountered both in the normal and in the bizarre stimulation (in flight) of the labyrinth. It may be linear such as is noted on take-off, landing, or on diving, or it may be angular which is more common in flying and

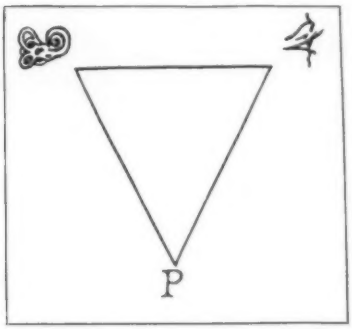

Fig. 1.—The equilibratory triad, representing diagramatically the relationship between the ocular, labyrinthine, and proprioceptive systems.

is encountered in banks, spirals, spins, recovery from spins, pull out from dives, and in most other aerobatic maneuvers.

Gravity—"G" is one of the important affectors of the labyrinth. The normal gravitational effect of the earth is operative with the flyer in the plane prior to takeoff. When he pulls out of a dive, hits an updraft, makes a tight spiral or a loop, he is subjected to increased positive "G." In the event of a downdraft, a roll, an outside loop, and several other maneuvers he is subjected to negative "G." That is, when in flight he is intermittently acted on by either more or less of the gravitational force of the earth than that which is exerted on a body at rest. It is important to note that centrifugal force cannot be distinguished from normal "G" force in most instances, nor can centripetal force be differentiated from "negative G."

The effect of these types of acceleration may be greatly modified by activity of the flyer himself. Suppose a man in a tight spiral, which is rapid enough to produce stimulation of his semicircular canals, looks quickly over his shoulder, that man has all the stimulation produced by the motion of the aircraft, with the motion of the head turning superimposed upon it. This can be a considerable amount and may well have a devastating effect upon his orientation. A graphic representation of the complex combination of types of motion may be seen in the accompanying diagram.

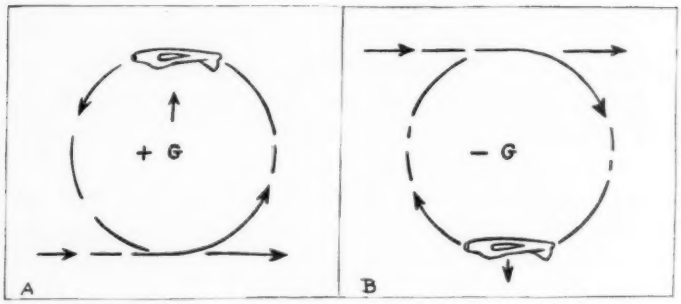

Fig. 2.—A, The normal loop, producing Positive G on the pilot. B, The outside loop, producing Negative G on the pilot.

EXAMPLES OF ILLUSIONS

Unperceived motion is not a specific illusion but gives rise to many illusions, by virtue of the fact that there is a definite threshold for changes in acceleration and direction above which motion must be made if it is to be perceived by the individual. In order to stimulate the semicircular canals, angular acceleration in excess of two degrees per second per second must be utilized. Acceleration at a rate below this value leaves the subject oblivious to the presence of any change at all. In the case of tilt, the average individual may be tilted 24 degrees upward or 10.6 degrees downward without being aware of any change in his position. It is as a result of this threshold that blind flight may well result in considerable irregularity of flight if the sensory organs are to be trusted entirely. The data given for threshold values of vertical acceleration vary between 4 and 12 centimeters per second per second. Those for linear acceleration vary between 2 and 20 centimeters per second per second (Armstrong[2]).

Unperceived Banks—In uncomplicated situations, tilting of one's body produces an irregular distribution of the pull of gravity which makes one realize that his body is tilted. In aircraft flight, however, the well coordinated turn is one in which the combination of gravitational and centrifugal force produce a vector acting at right angles to the transverse axis of the aircraft and leaving the flyer entirely unaware that any change in position has taken place, if the rate of change is below the critical values mentioned above.

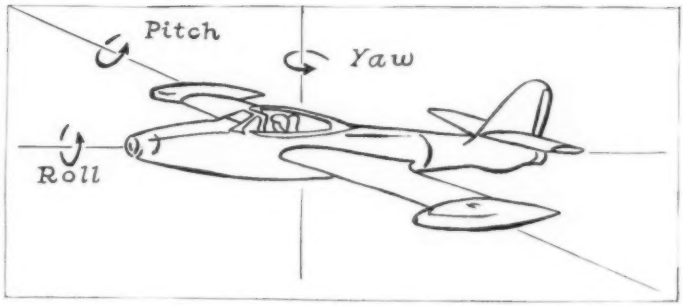

Fig. 3.—The axes of a plane showing diagramatically the separate types of motion which are possible in an aircraft. Any possible combination of these motions may, and at times does, occur.

Coriolis Reaction—Illusion—The Coriolis reaction is that sort of response to motion of an aircraft by which the vertigo produced by the aircraft is aggravated by means of motion made on the part of the flyer within the plane. Thus, a plane in a spin will cause the pilot to develop vertigo. If he moves his head to "sweep" the sky during this maneuver the vertigo which is produced may well be many times as severe as otherwise would be the case. In a more specific situation, if the plane is spinning down and the head is moved toward the floor of the cockpit the sensation may be produced that the plane is now diving or spinning beyond the vertical. The tendency to pull back on the controls, would, in a spin, merely aggravate the situation (Coriolis[11]).

Analysis: Predominant effect, semicircular canal. The explanation for this aggravation of the vertiginous effect may be readily seen if one will hark back to the fundamentals of labyrinthine physiology. In the first place, it is well known that the plane of vertigo is established at right angles to the axis of rotation. Furthermore, it is known that if the plane of vertigo is once established it will be maintained at the same relation to the head, even if the head position be changed. When this occurs, the objective vertigo is greatly aggravated and the pilot becomes ordinarily confused. Thus we imagine a plane in a spin with the pilot's head so that there is maximum stimulation of the horizontal canals. When the plane of vertigo is well

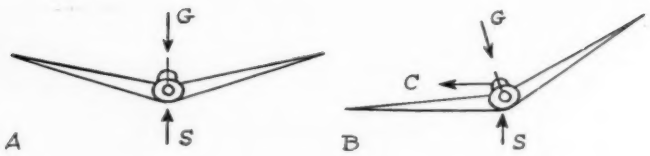

Fig. 4.—A, Sustaining Force and G Force represented in normal flight. B, Sustaining Force and G Force and their relation to centrifugal force in an aircraft flying in a circle.

established it is parallel to the horizontal canals. If his head was down and he is accommodated to the motion of spinning sidewise, he is suddenly confused when he sits up and finds the plane of the apparent rotation has shifted violently from perpendicular to horizontal. In the specific example of the pilot who moved his head to see the floor of the cockpit, we have an example of the confusion which arises when the plane of vertigo is suddenly changed. In this instance the change produces the erroneous sensation that the plane is now diving beyond the vertical.

The Leans—Illusion—This illusion may be produced in two distinct ways although the mechanism producing it is the same. In instrument flight, when visual orientation is impossible, the aviator feels a sharp rolling motion of the aircraft and is aware that he has been tilted out of horizontal by that motion. The aircraft is then gradually returned to a horizontal attitude and the flyer, although oftentimes believing the instruments, may remain uncomfortable and feel as if the plane is still tilted out of level flight. His natural reaction is to lean in the other direction to compensate for this out of horizontal attitude of the aircraft. The illusion may be brought about in exactly the opposite manner, that is by gradual tilting of the plane with sudden recovery giving the illusion to the flyer that he has been suddenly tilted out of his normal vertical position. He then leans to accommodate for it. This illusion is oftentimes relieved only when the plane is brought from instrument flight to visual flight and proper orientation may once more be made by means of the ocular system (Armstrong,[2] Kafka[12]).

Analysis: Predominant effect, semicricular canals. The sudden tilting of the aircraft stimulates the semicircular canals at the rate

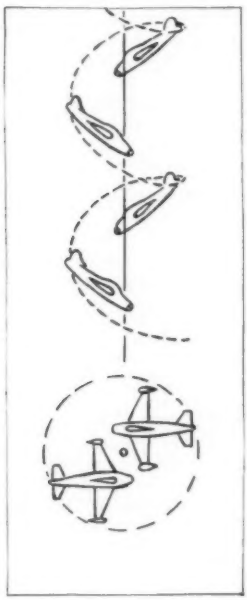

Fig. 5.—Aircraft in a spin. A, Lateral view. B, Top view. This demonstrates the violence of rotation produced by this maneuver.

of more than 2 degrees per second per second. This stimulation is above threshold value and the labyrinthine end organ notifies the central nervous system that the attitude of the flyer has been changed. Slow recovery from that attitude does not stimulate the semicircular canals in reverse direction and for that reason the impression that the attitude of the aircraft is incorrect remains uncorrected. It is for this reason that the lean of the flyer takes place and is not corrected by the correction of the position of the aircraft. The reverse situation produces the same result, slow tilt without stimulation followed by sudden recovery with stimulation above threshold levels and a resultant "lean."

Underestimating the Degree of Bank—Illusion—It is quite common for the pilot to underestimate the degree of bank in an air-

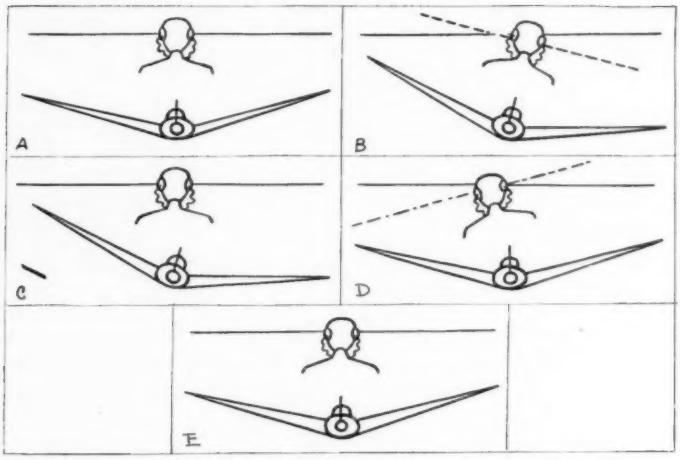

Fig. 6.—Demonstration of sequence of events in the "Leans." A, Normal flight. B, Updraft tips plane and pilot's illusory horizon. C, Pilot rights self and orients illusory horizon with the true horizon. D, Plane is slowly righted and illusory horizon is tipped away from true horizon without pilot being aware of change. E, Pilot sees reference object and correctly orients illusory horizon with true horizon.

craft and for that reason he banks too steeply on entering a turn and over-corrects on leaving a turn (Armstrong,[2] Aviation Otology[5]).

Analysis: Predominant mode of action, semicircular canals. Because much of the rotation of the aircraft in banking is below the normal threshold stimulation of the semicircular canals the pilot finds it necessary to turn the aircraft farther than actually desirable in order to produce the necessary stimulation of the labyrinth which notifies him of adequate banking. The same is true when he recovers from the turn. Much of his recovery from banking is below threshold stimulation and as a result he overcorrects the position of the aircraft before adequate stimulation of his labyrinth has occurred.

Reversal of Rotation — "The Graveyard Spin" — Illusion — In this situation the pilot is coming down in a spin or tight spiral without visual reference and levels the plane out. He feels that he has

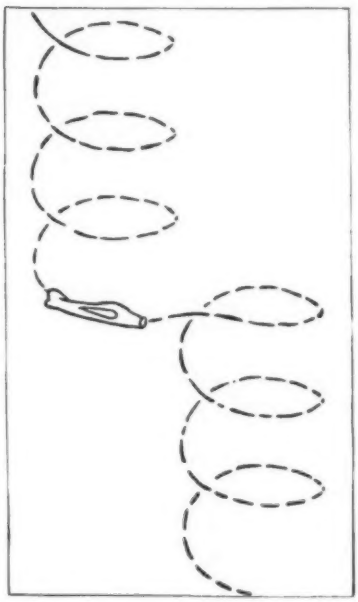

Fig. 7.—Plane in spin, comes down, levels out and then goes back into spin. This combination is known as the graveyard spin.

overcorrected for the spin and immediately puts the plane back into a spin in the same direction as before, where he rests perfectly comfortably, unaware that he is again spinning toward the ground (Armstrong,[2] Aviation Otology,[5] Northington[13]).

Analysis: Mode of action, semicircular canals. In rotation of the semicircular canals, if the rotation is maintained long enough to properly accelerate the movement of endolymphatic fluid, sudden cessation of that motion causes the endolymph to continue to flow in the opposite direction, giving the sensation to the person that turning is now occurring in the opposite direction. This is called reversal of rotation. Thus a pilot spinning to the left corrects to level flight but feels as if he has overcorrected and is now spinning to the right, due to the continued flow of his endolymph. He, there-

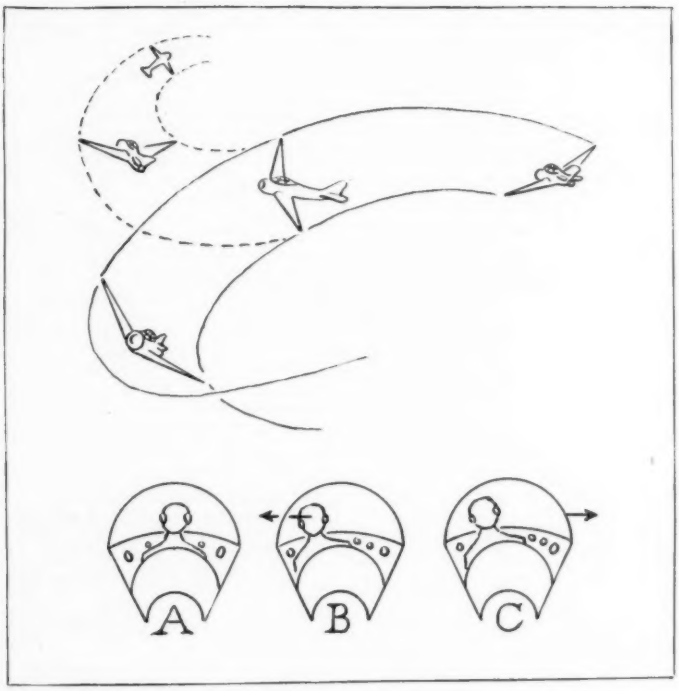

Fig. 8.—The sensation of opposite tilt in a skid. The solid lines represent the course of the aircraft and the dotted lines represent the direction of the illusory aircraft. A, Well coordinated turn. Pilot not thrown to either side of cockpit. B, Poorly coordinated turn to right. Pilot is thrown to left side of cockpit. C, Slipping turn to left. Left side of cockpit presses on pilot.

fore, puts the plane back into rotation to the left and feels perfectly comfortable that the plane is now in level flight and may well continue this attitude into the ground, without paying adequate attention to his instruments.

Opposite Tilt in a Skid—Illusion—The usual situation one notes when the aircraft skids on a turn is that his body is thrown to the inside of the aircraft during the process of a skid. That is, in a turn

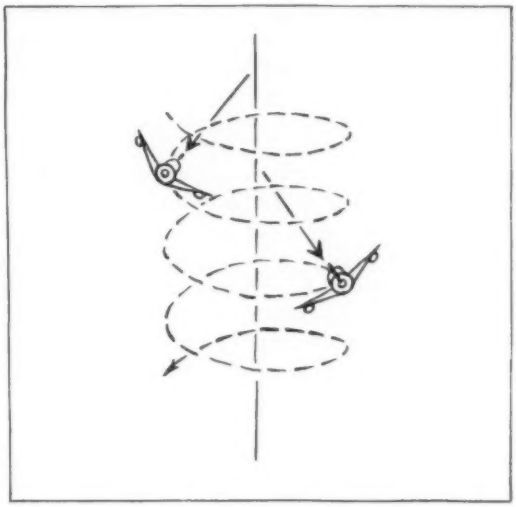

Fig. 9.—Arrow represents increased G force produced by tight spiral.

to the left with a skid to the right, he is pressed against the left side of the cockpit. The sensation which the flyer had, however, when being pressed to the left side of the cockpit is that the turn is to the right and that centrifugal force is pressing him against the outside of the cockpit, the left side in this instance. Thus, in a turn to the left, with a skid to the right he feels as if the plane were turning to the right in a poorly coordinated turn, with an improper amount of bank (Armstrong[2]).

Analysis: Predominant effect, proprioceptive system. When the aircraft skids in a turn, the centrifugal force throwing the pilot to the outside of the curve is overcome by slipping of the plane and the pressure of the cockpit is on the inside of the turn. In addition, this centrifugal force does not act at right angles to the transverse axis of the aircraft, so that the sensation is that the plane is banked in the opposite direction from that which it actually occupies.

Ascent on Descent in Tight Spiral—Illusion—In normal flight, when an aircraft is climbing there is an increase of G force pressing

the flyer into the seat. This is one of the common sensations in flight. When the pilot is flying under instrument conditions, in a tight spiral down, the pressure on the "seat of his pants" tells him he is under increased G force and he feels that he is climbing. It is in this situation that the pilot is unable to tell whether he is going up or down.

Analysis: Predominant effect, proprioceptive system. The "G" force in climbing is noticed on "the seat of the pants," by the stretching of the abdominal mesentary, by drooping of lids, lips, and cheeks. When the plane is in a tight spiral, centrifugal force throwing the pilot toward the floor of the cockpit reproduces all of these sensations and although the plane may be in rapid descent the centrifugal force may be sufficient to fool the flyer into believing that the plane is in ascent, a potentially fatal error of judgment.

Diving During Recovery from Turn—Illusion—In the course of diving an aircraft the pressure between "the seat of the pants" and the seat of the cockpit is less than in normal flight. Thus, the pilot who is recovering from a turn feels less pressure on "the seat of his pants" and is of the opinion that the plane is diving. To compensate for this situation he pulls back on the stick and elevates the nose of the plane from level flight to a steep climb and possibly a stall.

Analysis: Predominant effect derived from proprioceptive system. In diving, less than normal "G" is expended on the pilot in a direction perpendicular to the long axis of the aircraft. On recovery from a turn the same situation ensues and when the "G" force becomes reduced, the pilot feels that he is diving the aircraft and logically recovers by elevating the nose of the aircraft; but in this instance he pulls it from level flight to a climbing and possibly a stalling attitude.

Oculogyral Effect—Illusion—Although this effect may well be important in the course of aviational work, it can best be described by utilizing the laboratory situation for an accurate description of the phenomena (Mann,[14] Graybiel and Hupp[15]).

This is noted when a subject is seated in a rotating chair in a dark room and is fixing his attention continuously on a target which rotates with the chair. A few seconds after the chair has reached the point of constant acceleration the subject notes that the target

seems to be displaced in the direction of rotation. After 18 to 20 seconds the displacement becomes less and then the target is noted to lag behind the rotation. This lag continues until the rotation is stopped. If the turning chair is stopped abruptly, exactly the reverse effect is noted by the subject. At first the target is displaced in a direction away from the direction of rotation and then it gradually drifts back, eventually reversing its position with regard to the direction of rotation. In the case of rather rapid acceleration, or deceleration, the subject immediately notices an occilation of the target for a few seconds which subsides and the target then drifts, as noted above. It is easily imagined that a pilot, in night flight, when confronted with a spin or tight spiral might well experience these same sensations in endeavoring to visualize his instruments. The confusion and difficulty in seeing a moving target may well account for the difficulties of night flight and particularly of recovery from a spin.

Analysis: Predominant effect, semicircular canals. On angular acceleration of the semicircular canals in the turning chair, there is some lag of the endolymph which stimulates the crista. It is thought that the elasticity of the crista and the viscidity of the endolymphatic fluid at first causes this displacement to be corrected for and then almost immediately the rotation produces the cristal deflection again. Once more the elasticity of the system may cause return to normal or nearly normal position until the rotatory acceleration has reached the point of equilibrium at which time the constant displacement of the cupula accounts for the sustained drift of the target. The reversal of drift during constant rotation would then seem to be produced by a reversal of the "inertial action" of the endolymph.

Audiogyral Effect—Illusion—This effect is that seen when the auditory stimulus received by an observer after a period of angular acceleration, seems to be displaced in the direction of the rotation (Arnoult,[16] Clark and Graybiel[17]).

Analysis: Predominant effect, semicircular canals. This illusion, which arises after angular acceleration of the semicircular canals, seems to be the result of the subject's perception of rotation with subsequent constant error of localization of sound in the direction of the original rotation. The average error in experimental determinations was 17.° The lack of importance attached to the need for accurate sound

localization in reciprocating engine aircraft and in jet aircraft makes this of little importance as an illusory state.

SUMMARY

The problem of sensory illusions of flying may be resolved into a very general observation which is that these illusions are, in reality, not illusions at all. They are the normal responses of the equilibratory triad to unusual motions. They are the responses of an animal normally involved only in two dimensional motion, who has become active in a three dimensional medium, but whose equilibratory and orientative mechanisms are still involuntarily adjusted solely to two dimensional motion.

Man is by nature and by evolution a terrestrial animal, who has not as yet evolved the physiologic makeup to permit proper aerial equilibration. Because his labyrinthine organs of orientation and equilibration are developmentally those of an organism adapted to two dimensional travel, or to motion which also is regulated by the ocular and the proprioceptive systems, he is entirely without stability or reliability when he is in a medium of high-speed three dimensional movement, without reference to both ocular and proprioceptive systems. The fundamentals of labyrinthine physiology have been understood for years and these fundamentals need only be logically applied to the new situation to have an adequate explanation for the seemingly incongruous responses which are obtained. One of the important characteristics of the labyrinth is that stimulation of it, by motion, must proceed at a rate above a certain threshold value in order to cause any recognizable response at all. That aircraft move at rates both above and below this threshold value is a factor fundamental in producing many of the illusory states.

The importance of understanding these physiologic foibles is clearly seen when one realizes that the pilot may suddenly feel that his instruments are not functioning properly and attempt to rely upon his own unreliable vestibular apparatus. If he does, death is a likely result. He should be made to understand that the ear gives perfectly normal responses to the stimulation received in flight, but that the unusual nature of the stimulation makes these responses entirely unreliable so far as orientation is concerned. This is a lesson which the young pilot learns easily but one which comes hard to the veteran, "seat of the pants" aeronaut who has already developed fixed patterns of reaction to situations aloft.

CONCLUSIONS

1. Vestibular stimulation in aircraft produces the same effects that similar stimulation would produce under any other situation.

2. The illusory states produced by vestibular activity, while flying, are the result of capricious stimulation of the labyrinthine apparatus, with normal responses resulting from this sort of stimulus.

3. The sensory responses with regard to aerial orientation are not only inadequate, but they are absolutely unreliable and must be completely distrusted if safety is to be maintained in the air.

4. The only adequate devices for orientation in space are those mechanical devices regularly a part of aircraft equipment and designed to aid in flying blind.

5. For proper equilibration, two of the three systems, (ocular, labyrinthine, and proprioceptive) must be intact and functioning.

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Figures 2, 3, 4, 5, 8, 9 were modified from illustrations contained in Pope-Otis: Elements of Aeronautics, New York World Book Co., 1941.

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LXVIII

MULTIPLE AREAS OF NECROSIS AND ANAPLEROSIS THROUGHOUT THE TEMPORAL BONE

BASED ON THE HISTOLOGIC STUDY OF TWO CASES

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During the past few months we have, in the Otological Research Laboratory, studied serial sections of the temporal bones of 300 patients from the medical, neurosurgical and pediatric wards of the hospital, who have been autopsied during the past 25 years. Many types of disease are represented in this collection—syphilis, tuberculosis, anemias, leukemias, cerebral or generalized arteriosclerosis, pneumonia, nephritis, brain tumors, etc. In many of these patients the hearing acuity had been examined as carefully as possible with the speech audiometer, tuning forks and the pure tone audiometer. The temporal bones were removed whenever permission was obtained to open the skull, regardless of whether the patient's hearing had been found to be normal or impaired. Our object in studying these histologic sections was to acquaint ourselves with the normal and pathologic anatomy of the temporal bone and to try to correlate the hearing acuity, as shown by the audiograms and other tests, with the morbid structural changes in the middle and inner ear, with the general systemic disease and with the clinical history.

It was during this study that we chanced to come upon the two series of histologic sections described in this paper. In the diagrammatic drawing (Fig. 1) the location is shown of the areas of bone necrosis in the temporal bones. The histologic appearance of these areas in their various stages of formation and repair is shown in the photomicrographs. In order to determine whether or not these areas of bone absorption are in any way related to blood supply a detailed study was made of vessels supplying the various structures in the

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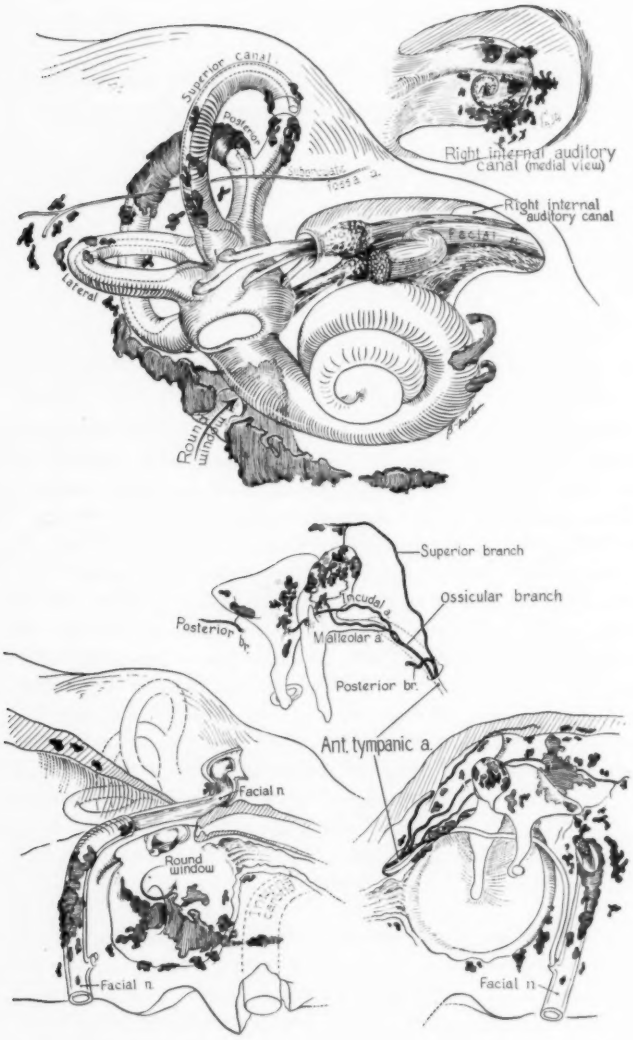

Fig. 1.—This group of diagrammatic sketches shows the general location of the osseous lesions in the temporal bone and ossicles of the two cases.

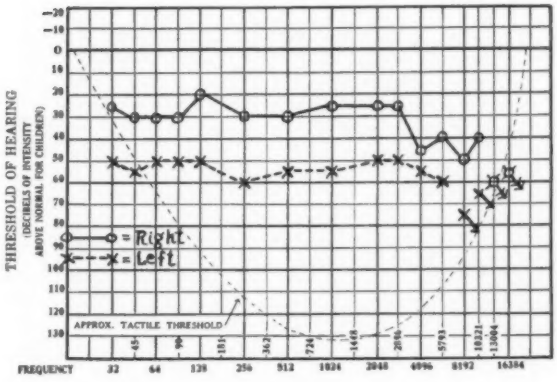

Fig. 2.—The audiograms, by air conduction, of Case 1.

middle ear, and especially the ossicles.[1, 2] Our conclusion is that the changes in the ossicles, shown in Figs. 4A, 4B and Fig. 5, and in other areas of the temporal bone are not due to interference with the blood supply.

CASE 1.—Path. No. 10692: This 49 year old white man had rheumatic fever and polyarthritis when he was 14 years of age. Ten years later he was admitted to the John Hopkins Hospital for cardiac decompensation. For the next 25 years there were several admissions for the same reason. During these periods of hospitalization it was noted that he had a penile scar, suspicious greyish white patches on the hard and soft palate and small, fixed pupils. Otherwise the neurological examination was negative. Serologic tests for syphilis were made on several occasions during this 25 year period, but were always negative. On Dec. 4, 1928 he was brought to the hospital with cardiac and renal decompensation and died four days later.

The last otologic examination was 14 months before death. The audiogram made at that time is shown in Fig. 2. The patient first noticed some bilateral hearing impairment about 1915, which has progressed during the past four years, especially on the left. Tinnitus in the left ear was constant but there was no history of otitis media, although for many years he had had atrophic rhinitis and a foul nasal discharge. He has never had vertigo. He heard the whispered voice on the right at 9 feet; on the left at 6 inches. Conversational voice 15 feet on the right and 20 inches on the left, with the opposite

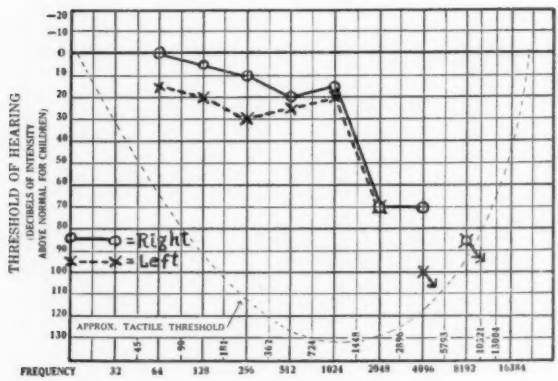

Fig. 3.—The audiograms, by air conduction, of Case 2.

ear masked. The 512 d.v. fork was heard better by air than by bone conduction on the right, but BC was better than AC on the left.

The post-mortem examination showed: (1) heart greatly enlarged, fibrous and calcified lesions of the mitral, aortic and tricuspid valves, extreme mitral stenosis and almost complete occlusion of the anterior descending branch of the left coronary artery; (2) chronic congestion of the viscera and lobar pneumonia; (3) no gross or microscopic evidence of syphilis was found.

The temporal bones were removed, fixed in 10 per cent formalin, decalcified in one per cent nitric acid, embedded in celloidin and sectioned serially in the vertical plane.

Case 2.—Path. No. 4018: This patient was a white male 41 years of age who was admitted to the Baltimore City Hospital in July, 1929 with cough for 5 months, pain in chest and loss of weight. The clinical diagnosis was advanced bronchogenic carcinoma with involvement of the left recurrent laryngeal nerve and secondary pulmonary infection. This was in accord with the history, physical findings and roentgen ray studies. The serologic test for syphilis was negative and sputum tests for tubercle bacilli were negative. A progressive febrile course terminated in death four months after admission.

The otologic examination was made in July, 1929, about four months before death. The patient did not complain of his hearing

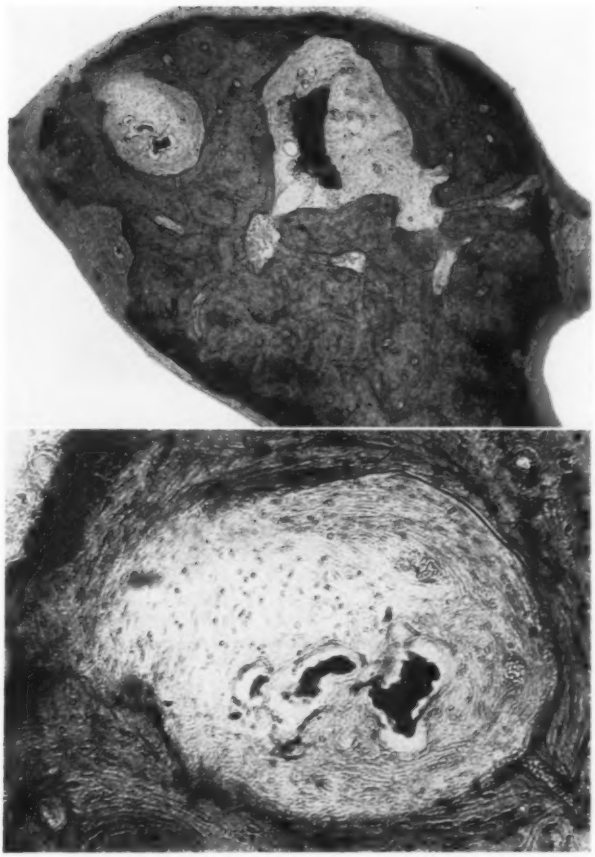

Fig. 4A.—Case 1, Right ear. A section through the head of the malleus at medium magnification. Two of the osseous lesions described in this paper are included in the area photographed. The smaller of the two lesions is shown at a higher magnification in Fig. 4B.

Fig. 4B.—See legend of Fig. 4A. The bony sequestra in this lesion are so darkly stained, in comparison to the other tissues, that their detailed structure does not show in the photograph.

Fig. 5.—Case 2, Right ear. Section through the head of the malleus, the body, the long process and the lenticular process of the incus, and the incudo-stapedial articulation. Note the extensive lesions of the malleus and incus, and the abnormal malleo-incudal articulation. The incudo-stapedial articulation is normal.

and had had no vertigo. The tympanic membranes were normal in appearance. A 512 d.v. tuning fork was heard better by air than by bone conduction on both sides, and BC was normal. The audiogram (Fig. 3) shows an abrupt loss of acuity for all tones above 1024 cps. Vestibular responses to cold caloric stimulation were normal.

The post-mortem examination showed: (1) squamous cell carcinoma in the left bronchus. Metastases to the right lung, the pericardium, the hilar lymph nodes, the liver, kidneys, suprarenals and spleen. The left recurrent nerve was involved in the tumor; (2) dilatation of the heart and chronic passive congestion of the lungs and abdominal viscera; (3) generalized arteriosclerosis; (4) nothing abnormal seen in the brain or meninges, and (5) no gross or microscopic evidence of syphilis was found.

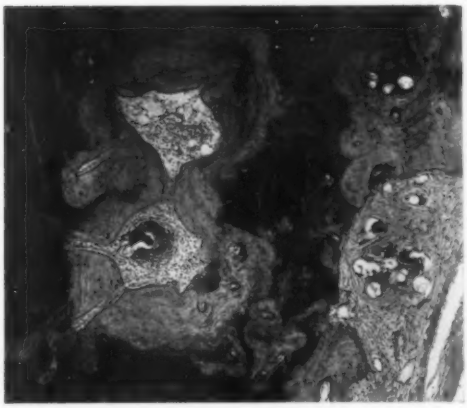

Fig. 6.—Case 1, Right ear. This lesion is in the otic capsule near the internal auditory canal. It shows a multinucleated giant-cell enclosing a central cavity which houses a bony fragment. Note to the right a group of cavities in the dura which are the sole residua of disintegrated former giant-cells.

The temporal bones were prepared in the usual manner and cut in the vertical plane. Every fifth section was stained with haematoxylin and eosin and available for study.

HISTOLOGIC STUDIES

The areas of bone absorption we are describing were bilateral in the tympanic, the mastoid and the petrous parts of the four temporal bones. Some of these areas were in the periosteal, some in the enchondral and others in the endosteal bone. The larger lesions may involve all three layers especially around the semicircular canals. Both of our patients died 25 years ago, one of mitral stenosis and the other of bronchogenic carcinoma. Neither had any symptoms suggesting bone disease and therefore no bone, other than the temporal bones, was taken for histologic study. The temporal bones of these two cases were removed as part of a long-time study to correlate hearing acuity, determined before death, with pathologic changes in the middle and inner ear and the acoustic nerve. Until these histologic sections were studied there was no suspicion that bone disease of any kind would be found.

Histologic study of these four temporal bones showed many lesions, varying in size and location, as shown in Fig. 1. The earliest

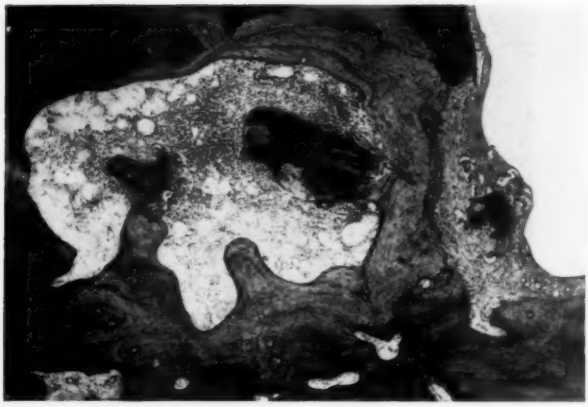

Fig. 7.—Case 1, Left ear. This lesion is near the round window. The photomicrograph shows cellular infiltration, sequestrum formation and healing with new bone formation in the periphery. A sequestrum is seen in the adjoining mucous membrane of the middle ear.

manifestation is necrosis of bone. As a result of it a granulation tissue appears with perivascular accumulation of round cells in localized areas. Then begins bone absorption and formation of sequestra. It is our impression that an unusual type of giant-cell plays a prominent part in the resorption of bone and in the destruction of sequestra. These giant-cells reveal a surprising variation in shape and size. They may have as many as a hundred nuclei distributed about the periphery of a central cavity (vacuole) in which frequently several types of breakdown-products of bone are present (Fig. 6). Osteoclastic and osteoblastic activity may appear simultaneously in the same lesion. In such areas one sees resorption of a sequestrum embedded in granulation tissue, while at the periphery of the granulation bed new bone is being laid down (Fig. 7). This new bone may then be invaded by ingrowing blood vessels and again undergo absorption. Some of these areas with repeated bone transformation may greatly resemble the so-called "Remaniement Pagétoïde" (Lièvre).[3] It is our impression that the bone disease we are describing is characterized by acute exacerbations, remissions, healing and occasional relapse.

In the external auditory canal, areas of bone necrosis are seen, predominantly in the floor of the canal. Here the superficial layers of bone were replaced with dense fibrous tissue, rich in cellular ele-

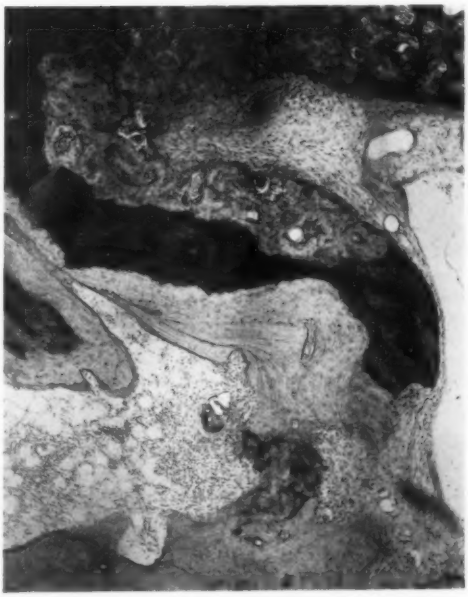

Fig. 8.—Case 1, Left ear. Typical areas of each of the two kinds of osseous lesions in this otic capsule are included in this medium power photomicrograph of a portion of the medial wall of the round window niche. The otosclerotic lesion is in the upper part of the picture, and several aspects of the lesion described in this paper can be seen in the lower half of the field. The two lesions are separated by a narrow layer of original otic capsule; it has irregular margins and is darkly stained.

ments. There is here little evidence of new bone formation. The overlying epidermis shows no signs of infiltration or ulceration. The eustachian tube was normal in Case 1, but in Case 2 a necrotic fragment of bone surrounded by cellular, vascular connective tissue is seen in the lateral osseous wall of the tympanic orifice on both the right and left side. Areas were seen in the lateral wall of the epitympanic space, in both Case 1 and Case 2, where bone had been resorbed and replaced with cellular granulation or dense fibrous tissue. It is precisely through this part of the epitympanic space that in Case 2 the malleolar and incudal arteries pass to supply the malleus and incus. The local pathologic process in the bone has not visibly involved these branches of the ossicular artery or interfered with the blood supply of the malleus and incus. In Case 1 the lesions in the ossicles

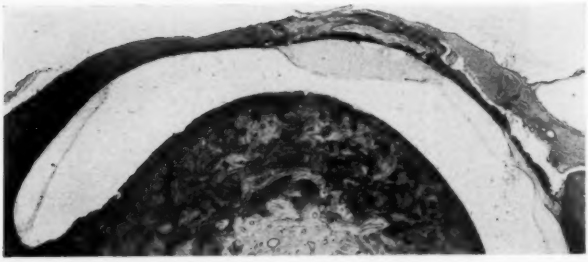

Fig. 9.—Case 2, Right ear. This low-power photomicrograph shows the extensive osseous lesion along the arch of the superior semicircular canal. The membraneous semicircular canal is seen on the extreme left and extreme right. Overlying the damaged bone is the dura. Underlying this damaged bone and protruding into the canal is young connective tissue, very poor in cells—evidently a reaction to the bone disease.

are circumscribed and contain sequestra (Figs. 4A and 4B). In Case 2 extensive areas of fibrous replacement, many containing small sequestra, are seen throughout the entire malleus and incus. The articulation between malleus and incus is involved, and in one place fibrous tissue has formed a fibrous synostosis (Fig. 5).

Although no clinical evidence of facial nerve involvement was noted in the neurological examination, histologic examination shows extensive areas of bone necrosis in the Fallopian canal (Fig. 1). Under these areas of bone destruction the perifacial fibrous tissue is thickened and protects the nerve. In Case 1 an area of typical otosclerosis is seen around the oval window. This condition was diagnosed before death by the history, appearance of tympanic membranes, audiogram and fork tests.[4] In Fig. 8 an area of otosclerosis and a healing bone lesion such as we are describing are seen side by side separated only by a small tongue of original labyrinthine capsule. The difference in their appearance is striking.

In the cochlea and semicircular canals the distribution of bone lesions is shown in Fig. 1. Histologically we have never seen involvement of endosteal bone in the cochlea. This is in marked contrast to the lesions in the semicircular canals where often the endosteal as well as the periosteal and enchondral bone is destroyed. In the specimen illustrated in Fig. 9 enchondral bone has been replaced with cellular, vascular connective tissue that has undermined extensive areas of endosteal bone, leaving them as sequestra. In the

Fig. 10.—Case 1, Left ear. Shows the osseous lesion in a section through the arch of the posterior semicircular canal. Note the sequestra and the large amount of dense fibrous tissue. The membranous canal is dislocated but otherwise intact.

region photographed in Fig. 10, as in the Fallopian canal, the fibrous tissue between the bone lesion and endolymph and perilymph spaces becomes greatly thickened as a protective measure. No new bone formation has been seen in this thickened fibrous tissue or in the membranous canals. Smaller lesions are seen in the osseous walls, where endosteal bone has been replaced with fibrous tissue, leaving an irregular bony wall.

Extensive lesions are seen in the floor of the internal auditory canal (Fig. 11). A typical area is shown in Fig. 12. The granulation tissue is vascular and very cellular, and the sequestrum is partially walled off with dense, hyaline fibrous tissue. Several layers of new bone surround the granulation tissue and demarcate the lesion from the normal bone of the otic capsule.

Fig. 11.—Case 1, Right ear. The left hand picture shows the location of lesions in the otic capsule near the anterior part of the basal turn of the cochlea and the lateral end of the internal auditory canal. The right hand picture shows one of the lesions in greater detail. Note the sequestra embedded in dense fibrous tissue and the newly formed bone at the periphery of the lesion.

DISCUSSION

The differential histologic diagnosis of these areas of bone necrosis, granulation tissue, sequestrum formation and healing with new bone formation and those bone lesions caused by late congenital syphilis is difficult but possible. In addition to the bone lesions described in this paper we see throughout the temporal bones of these two cases numerous collections of round cells, mostly lymphocytes and plasma cells. Occasionally these infiltrates have no relation to blood vessels, but usually they are perivascular. Such a perivascular accumulation is seen in Fig. 13. Here the vessel is in a bed of granulation tissue, but we have seen similar perivascular collections of lymphocytes and plasma cells in the tympanic membrane, in the middle ear mucosa, in the fibrous tissue that lines the jugular fossa, in the fibrous tissue

Fig. 12.—Case 1, Right ear. Detail of a typical lesion; this one is in the otic capsule near the internal auditory canal. Note the vascular, cellular granulation tissue and the sequestrum being walled off with dense fibrous tissue.

of the carotid canal, in Scarpa's ganglion, and in the cochlear nerve fibres in the osseous spiral lamina distal to the spiral ganglion. No polymorphonuclears or eosinophiles have been seen in these isolated or perivascular accumulations. Also no necrosis, caseation, epithelioid or giant-cells of Langhans' type have been seen. In papers by Otto Mayer of Vienna and J. S. Fraser of Edinburgh on the Pathological Changes in the Ear in Late Congenital Syphilis[5, 6, 7] the accumulation of lymphocytes, frequently arranged around the smaller blood vessels were regarded as true miliary gummata. In the histologic sections of the temporal bones of their cases they were able to trace all the intermediate stages from these collctions of lymphocytes to the classical miliary gumma with epithelioid cells, giant-cells and caseation.

Late congenital syphilis is one form of lues acquired prior to passage through the birth canal. Dependent on the duration of the infection in utero, the characteristics of the spirochetal strain, and the efficacy of maternal or fetal defense mechanisms, any of the following conditions may result: (1) Syphilis of the fetus; (2) early or infantile syphilis, or (3) late congenital syphilis. In late congenital syphilis the infant appears normal and frequently serologic tests are negative. The infection may not become apparent for

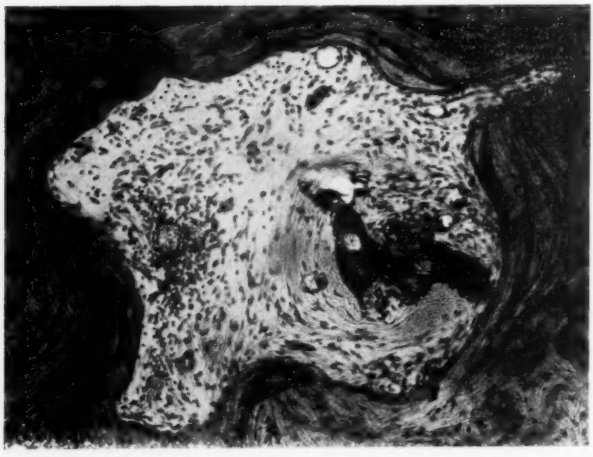

Fig. 13.—Case 1, Right ear. This area of bone necrosis is in the otic capsule near the internal auditory canal and shows accumulation of lymphocytes and plasma cells around a blood vessel, also a sequestrum to which are attached multinucleated giant-cells that seem to act as osteoclasts.

many years, but most patients exhibit symptoms during childhood or adolescence. The common clinical manifestations are interstitial keratitis and neurosyphilis. Certain stigmas, believed to be the result of spirochetal infection of cells in the embryo from which the affected structures arise, are characteristic of late congenital syphilis, such as Hutchinson's teeth, impaired hearing of the nerve type and a variety of skeletal changes.

Neither of our patients had eye lesions, defective teeth nor any history or post-mortem evidence of late congenital syphilis. They did, however, have impaired hearing or loss of acuity for high tones (Figs. 2 and 3). For comparison, detailed studies were made of serial sections of 13 temporal bones from 7 patients with clinically established lues who had similar impairment or loss of acuity for higher tones. The post-mortem examination proved in 3 of them the existence of a tertiary stage of syphilis (syphilis of the aorta or central nervous system). In 5 of these 7 patients the high-tone loss was of the gradual type, that is an increasing loss of acuity for every octave from the low to the high frequencies, due to atrophic changes in the cochlear nerve that supplies the basal turn. The cells of Corti's organ were not involved. In 2 patients the high-tone loss was of the

abrupt type. The hearing was approximately normal for all tones up to some one of the frequencies in the speech range, after which there was an abrupt and marked impairment or total loss of hearing for the next higher tone and for all the yet higher tones. In this latter group both the organ of Corti and the nerve in the lower basal turn are seriously damaged. The histologic lesions in the temporal bones of these patients with proven syphilis differ in no way from those seen in the ears of patients with similar high-tone loss, but with no clinical or pathologic evidence of syphilis.[8]

SUMMARY AND CONCLUSIONS

A histo-pathological study of two cases showing multiple bone lesions throughout the tympanic, the mastoid and the petrous portions of the temporal bone is presented. These areas of bone necrosis and replacement reveal a preference for certain regions in each part of the os temporale, particularly in the petrosa. Their characteristics are the co-existence of florid, healing and healed lesions, and the tendency to exacerbations, remissions, healing and occasional relapses.

It is our impression that these bone lesions do not originate from a localized vascular disease, nor do they result from an infarct. Great similarity exists between the histo-pathology of these two cases and seven hitherto known cases, of which five are published in Mayer and Fraser's monograph on Late Congenital Syphilis of the Ear.[7] Certain histological differences and the complete absence of clinical and post-mortem evidence for a possible luetic etiology, however lead one to suspect the presence of a hitherto unknown disease, which has much in common with late congenital syphilis, or that we are confronted with a syndrome of different etiologies.

Our conclusion is that the bone lesions described in this paper played no part in the causation of the hearing impairments in our two patients. The impairments of Case 1 are explained by otosclerotic lesions, and of Case 2 by atrophy of both the organ of Corti and the cochlear nerve supplying the basal turn of the cochlea. A detailed study of the temporal bones of Case 2, and many others with similar hearing impairment—some with serologic, clinical and pathological evidence of syphilis and others with no evidence of the disease—are given in a paper on the Pathology of High-Tone Deafness.[8] Furthermore there is no evidence that the areas of bone necrosis in these two cases caused clinical symptoms of any kind. The etiology is obscure, but the lesions are quite definite, and it is surprising that they have not caused clinical symptoms.

ESCHENHOF.

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LXIX

STUDIES OF THE ACOUSTIC REFLEX

PART I

ELECTROMYOGRAPHIC STUDIES OF THE ACOUSTIC-AURICULAR REFLEX

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The reflex movement of the auricle of the guinea pig, cat, or rabbit evoked by an acoustic stimulation is called the acoustic-auricular reflex. The response in the guinea pig is especially marked. This reflex is also called Preyer's reflex after its discoverer.

Since Preyer's[9] report on this reflex movement, this phenomenon has been studied from various standpoints. The following were prominent in these studies: Stepanow,[12] Marx,[8] and, in Japan, Hoshino,[4] Saito,[10] Satta,[11] Yoshida,[15] Kidokoro[7] and Yanagihara.[14]

The present authors have made observations on the action current of auricular muscle during reflex contraction. The purpose of this paper is to report on several problems involved interpreted through electromyographic studies.

EXPERIMENTAL METHODS

Twenty adult guinea pigs, 500-700 grams in weight, were used in this experiment. The animals were not restrained during the experiments. Electromyographic observations on the reflex movements of the antitragic muscle lying at the antitragic part of the auricle were made. This muscle was chosen because of its active reaction to the acoustic stimulation and because of the ease in recording the action current from this muscle as compared with the other auricular muscles. A co-axial needle electrode was used to lead the action

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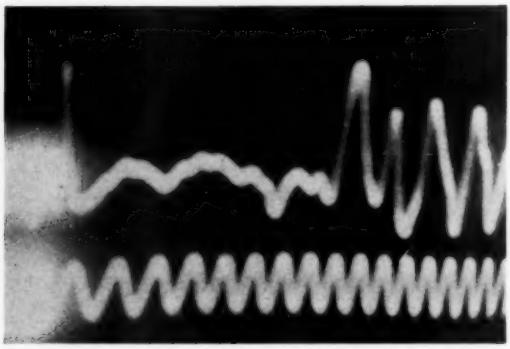

Fig. 1.—Electromyogram of the auricle muscle measuring the latency of the acoustic auricle reflex. Upper line: electromyogram; lower line: time mark (2000 cps).

current from the muscle. This method was suitable for observing the activity of either one or several of the neuromuscular units of the muscle.

Another method employed a photo-electric cell to record the onset of the reflex movement of the auricle. It recorded the change in electric current passing through the photo-electric cell as a result of the movement of the shadow of the auricle.

Two forms of apparatus for producing sound were utilized. One was a loud speaker connected to an audio-frequency oscillator. The other was an apparatus which consisted of an iron ball, 1 cm in diameter, to be dropped on an iron dish. This apparatus was used to generate sharp sound and was synchronized with the sweep of the oscilloscope.

RESULTS

a) *Measurement of Latency.* The time interval between the beginning of an auditory stimulus and the beginning of the action current in the auricular muscle caused by the auditory stimulation was recorded as the latency of the reflex. The distance from the sound source to the animal was 10 cm. The latency of the reflex was measured five times on each of five animals by the electromyographic method (Fig. 1). The results were: average latency 4.85 msec (milliseconds), the 95 per cent confidence limit 0.39, and the mean square 3.29. On the other hand, the latency as measured in

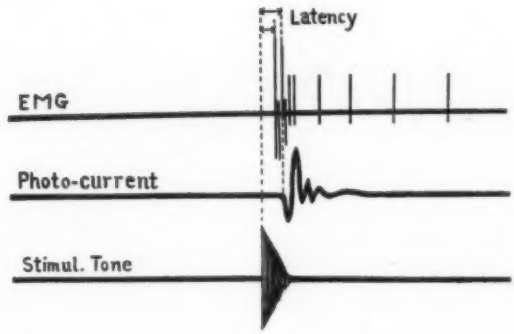

Fig. 2.—Schematic diagram showing the reflex time of the acoustic auricle reflex measured by the electromyographic and photo-cell method.

seventeen animals by the photo-electric cell method was: average 8.23 msec, the 95 per cent confidence limit 1.69, and the mean square 3.29. Therefore, the latency as measured by means of the electromyographic method was shorter than that as measured by the photo-electric cell method (Fig. 2).

The variations in the latency caused by change in the intensity of the stimulating sound were also measured. Sound intensity was controlled by varying the distance between the animal and the sound source. The latency was measured at four different distances (10 cm, 30 cm, 60 cm, and 1 m) five times on each of the five animals. The variation in the latency produced by the different intensities of the stimulating sound was greater than the variation to be expected resulting from the individual differences in the animals. Therefore, it is permissible to say that there was significant difference in the latency of the reflex at the four intensities of stimulation (according to the statistical level of 99 per cent significance). Incidentally, the reflex movement was not observed if the sound source was set at a distance of 1 m or more from the animal.

The results of these measurements may be summarized as follows: the latency of the acoustic-auricular reflex as measured by the electromyographic method had a maximum value of 6.94 msec and a minimum value of 4.85 msec and changed inversely as the intensity of the stimulation.

b) *The Electromyogram and the Movement of the Muscle.* In order to observe the action current of single neuromuscular units

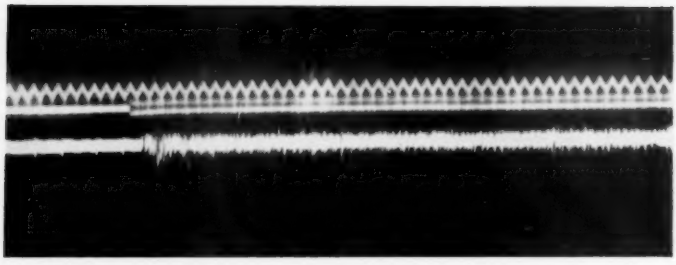

Fig. 3.—Oscillographic record of the electromyogram in tonic neuromuscular unit of the auricle muscle. Upper line: time mark (100 cps); middle line: stimulating sound; lower line: electromyogram.

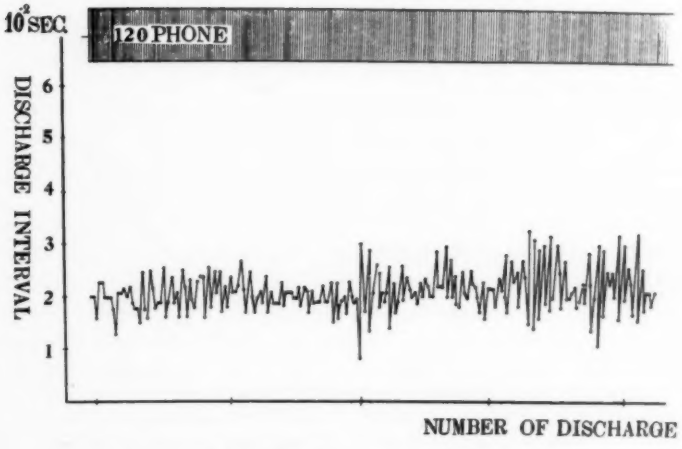

Fig. 4.—Interval diagram of the spike discharge in the tonic neuromuscular unit stimulated by the continuous sound.

of the auricular muscle, a co-axial needle electrode was inserted into it. The neuromuscular units of this muscle were classified into two types according to their mode of action. The first unit, type A, reacted with a spike discharge of 0.3 mV or less in electrical potential with a discharge interval of 20 msec. (A few of the units of type A discharged spontaneously without any stimulation with an interval of about 60 msec (Fig. 3, 4). When the animal was stimulated with sounds of gradually increasing intensity, almost all type A unit began to discharge when the intensity reached 90-100 phones. As the intensity of the stimulation increased, the discharge interval became shorter, but it did not become shorter than 10 msec (Fig. 5). Between the onset of the discharge and the time of the minimal discharge interval there was a difference in the sound intensity of five phones. When the sound was gradually decreased in intensity, the reaction in type A units was the inverse of that occurring when the sound intensity increased. Most units ceased their activity at 90-100 phones, but a few units continued in spontaneous activity. When the animal was stimulated by a sound of more than 90 phones in intensity, the type A units reacted with a discharge interval suitable to the stimulation. There was no "adaptation of the activity" (see next paragraph) in type A units. Thus, the interval of discharge remained the same during continuous sound.

The second type unit, type B, had a higher electrical potential (0.5 mV) than that of the type A unit. The minimal discharge interval was 5 msec. The minimal threshold of the reaction was 110 phones (Fig. 6). The difference in the intensity of the stimulating sound at threshold and at the moment of minimal discharge interval was five phones. If the intensity of the stimulating sound was sufficient in loudness or was suddenly increased in loudness, the reaction of minimal discharge interval began soon after the onset of the stimulation. Upon continuous stimulation with sound of the same intensity, the discharge interval became gradually longer until the discharge ceased several seconds after the beginning of the reaction. This phenomenon is called "adaptation of the activity." (Fig. 7) At the end of the stimulation the reaction of type B units ceased promptly and left no after discharge, nor was there any spontaneous discharge.

There was a close relationship between the activities of both types of unit and the movement of the auricle. After a weak stimulation or at the beginning of a gradually increasing stimulation, type A units were activated and the auricular muscles contracted weakly. When the discharge interval of this unit became shorter and the number of acting units increased, the tone of the auricu-

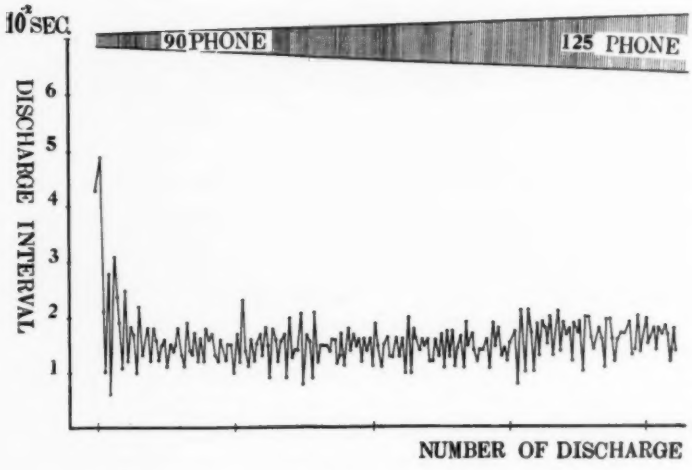

Fig. 5.—Interval diagram of the spike discharge in the tonic neuromuscular unit stimulated by the gradually increasing sound.

lar muscle became strong and the auricle was pulled gradually backward. Upon sufficiently strong stimulation, the type B units became active so that the muscle contracted spasmodically and the auricle was pulled rapidly backward. At the same time, the external auditory meatus became narrow from contraction of the perimeatal muscle. Proportionate to the adaptation occurring in the activity of the type B units, the contraction of the auricular muscle relaxed and the auricle gradually became erect. At this time type A unit activity still continued. With the termination of the stimulation, the activity of both units stopped—except for the spontaneous discharge of a few type A units—and then the auricle became completely erect.

Summarized briefly, type A units with a low threshold and no adaptative activity controlled the tone of the auricular muscle. Type B units with a high threshold and significant adaptative activity controlled the active movement of the muscle. These two types of units co-ordinate with each other in the reflex activity of the auricular muscle.

DISCUSSION

a) *Latency.* The latency of the reflex, i.e., the time interval between the reception of the stimualting sound and the reactive con-

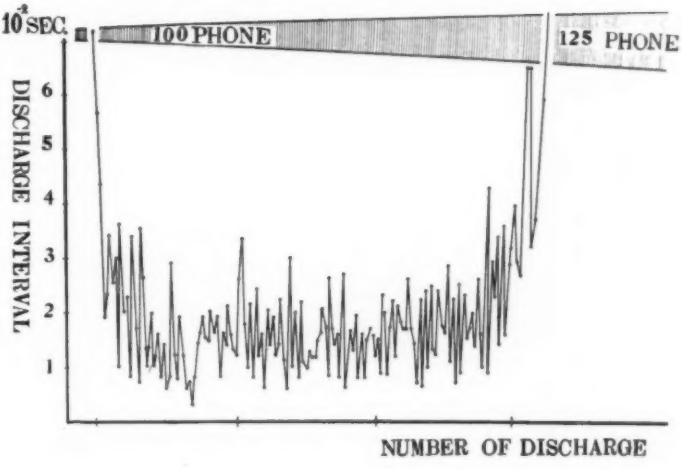

Fig. 6.—Interval diagram of the spike discharge in the kinetic neuromuscular unit stimulated by the gradually increasing sound.

traction of the auricular muscle, is the key to the reflex arc. In 1929 Satta[11] investigated the latency of this reflex and reported that its latency was the shortest of all the reflexes reported up to that time. Therefore, a simple pathway may serve in this reflex. As determined electromyographically, the time interval between the reception of the acoustic stimulation and the beginning of the reactive action current in the auricular muscle was 4.85 msec. Hoshino[4] observed the reflex in animals after various transsections of the midbrain and concluded a supposed reflex arc. This reflex resembled that of the acoustic reflex of the tensor tympani muscle as reported by Lorente de Nó, because the latency of both reflexes was almost the same.

Derbyshire and Davis, Kemp, Coppee and Robinson,[6] and Ades[1] measured the time required for nerve impulses to reach various levels of the auditory pathway. The latency of the electrical response to acoustic stimulation was 0.1 msec or less at the cochlea, 0.7 msec at the cochlear nerve, 1.6-1.75 msec at the trapezoid body, 2.5-3.5 msec at the superior olivary complex, and about 3.6 msec at the lateral lemniscus. These values give indication of the number of neurons involved in conducting the impulse between the cochlear nucleus and the facial nucleus. Thus, on the basis of these values for the latency of this reflex at different levels in its neural pathway,

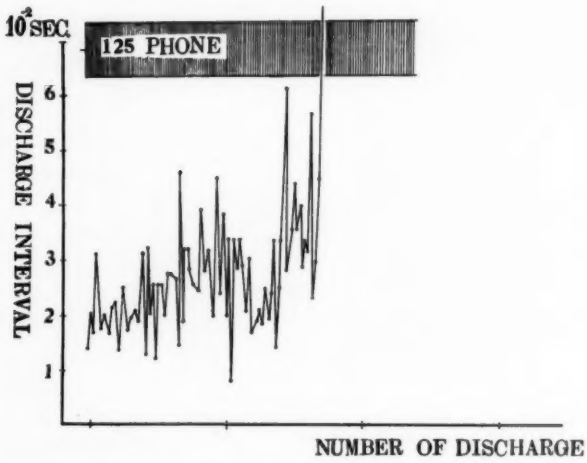

Fig. 7.—Interval diagram of the spike discharge in the kinetic neuromuscular unit stimulated by the continuous sound.

and allowing for an almost negligible conduction time in the nerve, and 0.5 msec for transmission across a muscle end-plate, the conduction time through the interneurons was calculated as follows: the conduction time through the interneurons is the remaining of the reflex time when the conduction time on the sensory pathway and the motor pathway are reduced. Allowing 0.7 msec for conduction on the sensory pathway and 0.5 msec for the same on the motor pathway, the conduction time through the interneurons is $4.85 - 0.7 - 0.5 = 3.65$ (msec). Therefore, allowing 0.7 msec for each synaptic transmission, there must be five synapsis (in other words four interneurons) involved namely, the cochlear nucleus, the superior olivary complex, the nucleus of the lateral lemniscus, and the facial nucleus. The reflex arc of the auricular reflex consists of: "cochlea—cochlea nerve—cochlear nucleus—superior olivary complex—nucleus of the lateral lemniscus—facial nucleus—facial nerve—muscle of auricle" (Fig. 8).

There are various kinds of functional deformations of nerve impulse crossing the synapses. For example, the time required for the nerve impulse to cross the synapsis, i.e. the "synaptic delay," is not the same in all instances, but is determined by the character of the impulse, especially its strength. According to the measurements noted

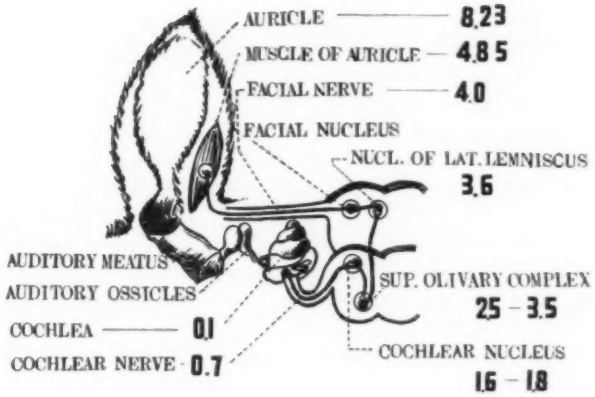

Fig. 8.—Schematic diagram showing the reflex arc and the transmission time (msec) of the acoustic auricle reflex.

in the experiments above, the latency of the auricular reflex caused by a loud sound was shorter than that caused by a weak sound. This latency difference is due to the difference in time required for passage of the impulse through the several synapses in the reflex arc.

The onset of the action current in the muscle indicated the beginning of the contraction of the muscle, but the onset of the actual movement was a few milliseconds later. This delay is physical and is referred to as the "activation time" by Sandow. The experimental difference of 4 msec in the latency as measured by the electromyographic method and that as measured by the photo-electric cell method corresponds to this "activation time."

b) *Nature of the Reflex.* These electromyographic observation revealed that the reflex movement of the auricle is caused by the reactive contraction of two kinds of muscle fibers coordinating with each other. The fibers which produce the muscle tone react to relatively weak stimulation and the fibers which cause the kinetic movement of the muscle react to stronger stimulation. In 1950 Tokizane[13] reported electromyographic studies of various muscles and showed that neuromuscular units of two types effect the contraction of these muscles. These units are the tonic and the kinetic neuromuscular units. The above mentioned neuromuscular units of the auricular muscle, designated as types A and B, correspond to these two units. Type A unit corresponds to the tonic unit and type B unit to the

kinetic. The total activities of these two units result in the contraction of the auricular muscle.

The reflexes of the body generally are divided into these same two groups according to the type of movement involved. The tonic reflex group is exemplified by the tonic neck reflex and the kinetic reflex group by the knee jerk. The reason for our special interest in the study of the acoustic-auricular reflex was that both tonic and kinetic activity are involved in this reflex. Also, each component is activated according to the nature of the stimulation, and the total of these activities causes the actual movement of the auricular muscle as the effector. Previous studies of the acoustic-auricular reflex lay no emphasis on the activity of the tonic unit because these studies concerned the reflex movements due chiefly to sharp sound. This present report is the first analytical study on the character of the complete auricular reflex.

CONCLUSIONS

Electromyographic studies of the acoustic-auriclar reflex in the guinea pig resulted in the following conclusions:

1) The latency of the reflex (reflex time) was 4.85 msec. With weak stimulating sounds it increased up to 6.88 msec.

2) The pathway of this reflex is presumably: "cochlea—cochlear nerve—cochlear nucleus—superior olivary complex—nucleus of the lateral lemniscus—facial nucleus—facial nerve—muscle of auricle." The magnitude of the latency of this reflex conforms to this scheme.

3) The acoustic-auricular reflex and the tensor tympani reflex have similarities in their pathway and latency.

4) The muscles of the auricle are made up of two types of neuro-musclar units—tonic and kinetic. The tonic unit, having a lower threshold and no adaptation, contracts slowly giving tone to the muscle; and the kinetic unit, with higher threshold and marked adaptation, contracts rapidly resulting in kinetic movement of the muscle.

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STUDIES OF THE ACOUSTIC REFLEX

PART II

EXPERIMENTAL STUDIES ON THE FUNCTION OF THE TENSOR TYMPANI MUSCLE

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The purpose of this article is to report on experimental work concerning the reflex contraction of the tensor tympani muscle to acoustic stimulation. The planning and execution of our experiments was greatly facilitated by studies of various other investigators in acoustic science and other fields of physics. Also, recent progress and improvements in experimental methods enabled us to design analytical experiments to investigate the nature of this reflex movement. Such investigations have previously been technically difficult. However, development of an electrical sound generator provided a more accurate source of acoustic stimulation than that previously used. Likewise, we were able to detect with ease the weak reflex contraction of the tensor tympani muscle by employing the electromyograph. Through its use the action of the muscle was observed directly as a pattern of the action current of its neuromuscular units.

The function of the two intrinsic muscles in the tympanic cavity has been postulated by many investigators. Helmholtz[3] was the first to suppose the function of the tensor tympani muscle to be concerned in stretching the tympanic membrane. Politzer's[12] opinion was that the reflex contraction of this muscle protected the inner ear against excessively loud sound. The protection theory stated that the two intrinsic muscles contracted in response to harmfully loud sound and thereby increased the acoustic impedance of the sound conducting apparatus. As a result, the very delicate structure of the inner ear was protected against acoustic trauma. Opposing this theory, Koehler advocated the "accommodation" theory, stating that the muscles of the middle ear tuned up the sound conducting system to implement

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the stimulating sound in sending vibrations of suitable intensity into the inner ear. In the present study we have tested the validity of both of these classic theories and have studied the true function of the tensor tympani muscle by using electromyographic analysis of its behavior to acoustic stimulation.

THE ELECTROMYOGRAM OF THE TENSOR TYMPANI MUSCLE

Experimental Methods. Sodium ouropane was injected into the femoral vein of a cat to produce slight narcosis. Then the tympanic bulla was opened from the ventral side. By means of two needle electrodes inserted into the muscle, the action current of the tensor tympani muscle was recorded. The sound generating apparatus used was the same as that used in the study of the acoustic-auricular reflex in Part I.

Results. Stimulation with a pure loud tone of 1000 cps evoked a series of spike discharges from the tensor tympani muscle at fairly regular interval. The neuromuscular units of the tensor tympani muscle were divided into two groups according to their electrodischarge behavior. This classification was similar to that used in the discussion relating to the auricular muscle. Thus, in this case the tonic neuromuscular unit, which discharged with low voltage and with a minimal discharge interval of 60-80 msec, showed no adaptation to continuous stimulation, while the kinetic unit, which discharged with a minimal interval of 40-50 msec and with high voltage, showed marked adaptation to continuous stimulation.

Upon stimulation with a pure sound of 1000 cps which was gradually increased in loudness, the spike discharges of the tonic unit were first observed at an intensity of 80 phones. The discharge interval became shorter upon increasing the intensity of the stimulating sound-up to the minimal discharge interval. The kinetic unit instituted its discharge only upon stimulation with an intensity of over 90 phones. However, its discharge interval gradually lengthened in the presence of a constant stimulating intensity until the discharge finally disappeared.

The muscle reacted to sudden stimulation by a loud sound with a synchronous discharge, i.e., all the units in the muscle discharged at the same time with almost constant interval. This interval was 60 msec and showed no adaptation to continuous stimulation (Fig. 9).

The above experiments were performed on the acoustically stimulated ear. In order to examine the behavior of the contralateral or unstimulated ear, the stimulations were applied to one ear after destroying the inner ear of the opposite ear whose tensor muscle was

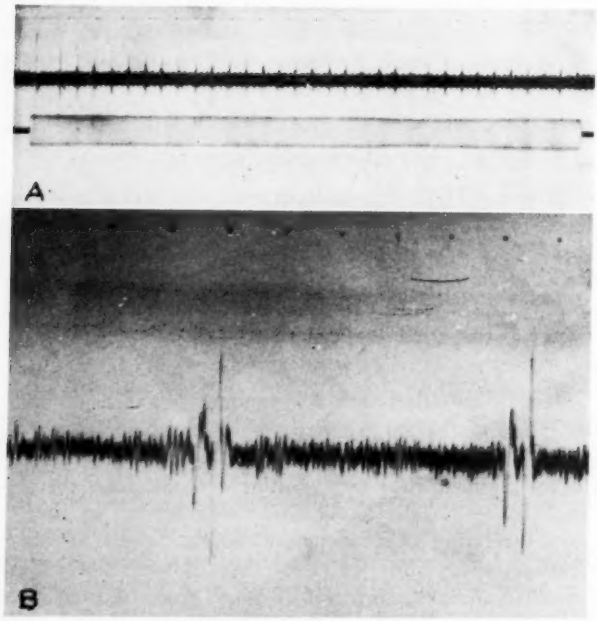

Fig. 9.—Oscillographic record of the grouped spike discharge of the tensor tympani muscle caused by the acoustic stimulation. a) Slow recording; b) rapid recording; time mark, 10 msec.

to be tested. Under these conditions the activity of the tensor tympani muscle as examined electromyographically in the unstimulated ear was almost the same as that in the opposite, intact, accoustically stimulated ear.

To examine the influence of narcosis upon the activity of the tensor muscle, electromyographic tests under various stages of narcosis were performed. The spike discharge of the tensor tympani muscle was not evoked in deep narcosis in spite of stimulation by a sufficiently loud sound. Following recovery from narcosis, the muscle activity recovered gradually. The electromyogram of the tensor tympani muscle observed following recovery of the animal from the narcosis was normal.

The spike discharge evoked by a sharp sound occurred 6-9 msec after the acoustic stimulation. This interval represented the latency

of the reactive contraction of the tensor tympani muscle to acoustic stimulation.

THE EFFECT OF MUSCLE CONTRACTION ON THE TYMPANIC MEMBRANE

Experiment Methods. The movement of the tympanic membrane caused by the reactive contraction of the tensor tympani muscle to acoustic stimulation was observed optically by noting the movement of a light beam reflected from a tiny mirror placed on the surface of the tympanic membrane. The light beam was sufficiently magnified to facilitate observation.

Observation of the isotonic contraction of the tensor tympani muscle was performed also by this optical method.

Results. Stimulation with 1000 cps pure sound of 120 phones in intensity caused a prompt inward movement of the tympanic membrane. The retracted tympanic membrane changed its position slightly at a rate of 15 times per second. When the animal was stimulated by a sound of increasing loudness, inward movement of the tympanic membrane was first observed at an intensity of about 80 phones. The inward movement of the drum gradually increased until the maximal retraction occurred at an intensity of the stimulating sound of about 90 phones.

When stimulation was by a sharp sound, the inward movement of the tympanic membrane stated 18-19 msec after the stimulation, and the drum reached its maximal retraction after 60 msec. Recovery occurred after 150 msec (Fig. 10).

Observations on the isotonic contraction of the tensor tympani muscle under a load of 130 mg in weight indicated the latency of the contraction to be 7-10 msec, but under a load of 5000 mg weight, contraction of the tensor muscle was not observed.

Continuous stimulation with a sound of constant strong intensity caused inward movement of the tympanic membrane nearly paralleling the intensity of the stimulating sound. At the end of the stimulation the displaced tympanic membrane did not return immediately to a normal position, but required about one second to do so.

DISCUSSION

Latency. The tensor tympani muscle contracts reactively to acoustic stimulation after a certain latent period. This latency was reported as 40 msec by Kato[4] and 10-20 msec by Lorente de Nó.[7]

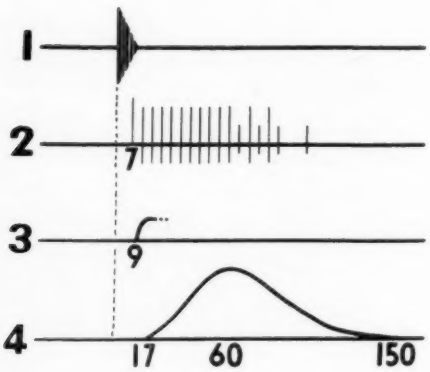

Fig. 10.—Schematic diagram showing the latencies (msec) of the tensor tympani reflex recorded by various methods. First line: stimulating sound; second line: electromyographic method; third line: contraction of the tensor tympani muscle; fourth line: retraction of the tympanic membrane.

By means of our electromyographical measurements the time interval from the onset of the stimulating sound to the reactive burst of the action current of the tensor tympani muscle was noted to be 6-9 msec, if a sufficiently intense stimulus were used. The latent interval was longer if a weaker stimulus were used. This latent period was almost the same as that recorded for the acustic-auricular reflex as noted in Part I. Accordingly, the number of synapses involved in the tensor tympani reflex arc may be the same as that involved in the acoustic-auricular reflex.

Furthermore, the latency of the isotonic contraction of the tensor tympani muscle was measured at 7-10 msec by the optical method. Therefore, the activation time of the muscle, following the onset of the action current, was about 2 msec. Moreover, 8-9 msec were observed to elapse following the onset of the contraction of the muscle before an actual effect on the tympanic membrane was noted. This delay occurred because the load on the tensor tympani muscle was considerably heavier than were the dimensions of the muscle. The factors impeding the effect of the contraction of the tensor muscle on the tympanic membrane are, presumably, the mass of the tympanic membrane and the ossicles, the tension of the ligaments connecting the auditory ossicles to the walls of the tympanic cavity, and the resistance of the labyrinthine pressure (Fig. 11).

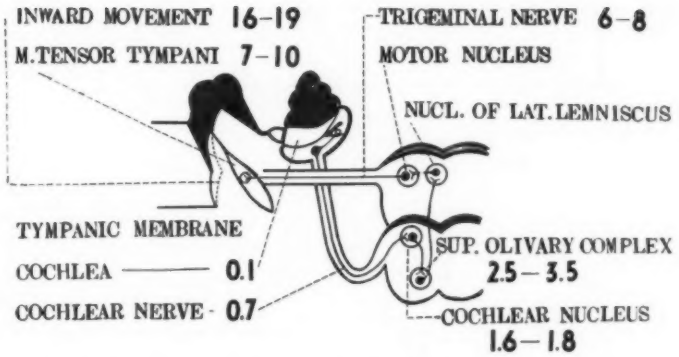

Fig. 11.—Schematic diagram showing the reflex arc and transmission time (msec) of the tensor tympani reflex.

Threshold. The tensor tympani muscle did not react to weak acoustic stimulation, but only to relatively loud sound. Kobrak[5] and Lorente de Nó[7] studied the reflex contraction of this muscle in cats and rabbits and reported that the threshold was considerably higher than was the threshold in humans. The threshold measured was 90 phones according to Kobrak's study, and 50-60 phones according to Lorente de Nó's. According to our optical observations, the threshold measured for pure sound of 1000 cps was 80 phones. The louder the sound stimulation, the greater was the inward displacement of the tympanic membrane caused by the contraction of the tensor tympani muscle. The fibers which reacted to weak sound near threshold belonged to the tonic neuromuscular units. The intensity of the stimulating sound required to produce reactive contraction of the kinetic neuromuscular units was greater than that required by the tonic units and was measured at about 90 phones.

Fluctuation of Muscle Tone During Contraction. Under sufficiently strong stimulation the tympanic membrane was pulled inward by the contraction of the tensor tympani muscle. It then remained in a retracted position throughout the duration of the stimulation. But under some conditions the position of the retracted tympanic membrane fluctuated slightly at a frequency of 15-20 times per second. This fluctuation has been noted by many investigators. Filehne,[1] Mangold and Eckstein,[9] and Wagner[14] supposed that this phenomenon was due to tetanic contraction of the muscle. Lorente de Nó[8] considered this phenomenon related to fluctuation in

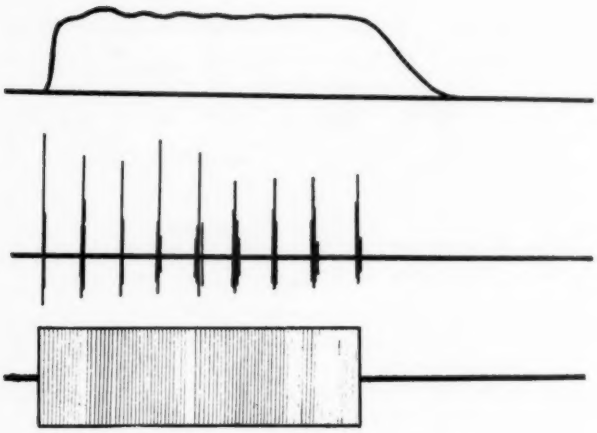

Fig. 12.—Schematic diagram showing retraction of the tympanic membrane caused by synchronous discharge of the tensor tympani muscle. First line: movement of the tympanic membrane; second line: synchronous discharge in the tensor tympani muscle; third line: stimulating sound.

the excitability of the primary auditory nucleus. We also observed this phenomenon in the records made by the optical method. The synchronous discharge observed on the electromyogram is brought to mind. The discharge interval of this synchronous discharge was about 60 msec, and the discharge bursts occurred about 16 times per second. Therefore, it is supposed that this fluctuation in the position of the displaced drumhead is related to the synchronous discharges of the tympanic muscle (Fig. 12). The cause of the synchronous discharge is not clear, but it is supposed not to be related to narcosis.

Bilateral Connections in the Reflex Arc. According to the studies reported by Hammerschlag,[2] Mangold and Eckstein[9] and Kato,[4] the reflex contraction of the tensor tympani muscle on both sides may be evoked by stimulating only one ear. The same phenomenon was observed in our studies both by the electromyographic and the optical methods. In other words, the action current of the tensor tympani muscle and the inward movement of the tympanic membrane were observed following acoustic stimulation of the contralateral ear. Furthermore, as to the latency and the strength of the reaction, there was no remarkable difference whether the reflex movements were evoked by stimulation of one or both ears. These ob-

servations showed that the afferent pathways of the reflex arcs from both ears joined each other in the reflex center, and the reflex center provided the efferent pathway to the tensor tympani muscles of both sides.

The Significance of the Tensor Tympani Reflex. From the above experiments we were unable to arrive at a final conclusion as to the significance of the tensor tympani reflex. There are various theories propounded by many investigators and these theories are divided into two groups: (a) the "protection" theory, according to which the inner ear is protected from excessive acoustic stimulation by changes in the acoustic impedance of the tympanic membrane as a result of reflex contraction of the tensor tympani muscle; (b) the "accommodation" theory, according to the vibration of the sound is accommodated by means of the tensor tympani reflex, so that a suitable amount of energy for perception by the inner ear is conducted through the drum of the middle ear.

The change of impedance necessary to prevent excessive vibration of the sound conducting apparatus must start at the same time as the acoustic stimulation itself if the protective function against excessive acoustic stimulation is to be fulfilled. However, these experiments have shown that there is a delay of a few milliseconds as a latency in the reflex. Thus, the inner ear cannot be protected against such loud sharp sounds as explosion or gun-fire.

On exposure to a continuous loud sound (as from a siren) or a succession of sharp sounds (as from a pneumatic hammer), the tensor tympani muscle continues its contraction and protects the inner ear from such acoustic violence. The decrease in acoustic energy caused by the increase of impedance secondary to the reflex contraction of the tensor tympani muscle was measured by means of cochlear microphonics by Wever and Bray[15] and Wiggers.[16] Their results indicated that such impedance caused a remarkable decrease (from 10-40 decibels) in the acoustic energy of low sounds (those below 1000 cps). However, since the acoustic energy was only reduced maximally by 40 db, the tensor tympani muscle cannot be expected to function as perfect protection against excessively loud sounds.

It would seem that the change of impedance on the tympanic membrane caused by contraction of the tensor tympani muscle should parallel the intensity of the stimulation. A similar situation is observed in the case of the iridic muscle of the eye. However, according to our electromyographic analysis of the contraction of the tensor tympani muscle, the threshold of contraction of this muscle is too high

to be affected by the sounds which are normal to everyday life. The amount of contraction noted was not usually directly proportionate to the acoustic stimulation as there was an even adaptative relaxation to continuous stimulation. Thus, we recognize the accommodative function of the tensor tympani muscle to stimulation, but this function is not as delicate and complete as that in the iridic muscles.

Summarized briefly, we can conclude nothing decisive as to the significance of the tensor tympani reflex, but we are able to appreciate both its incomplete function of protection against acoustic violence and its gross function of accommodation to acoustic stimulation.

We may expect further study on this problem from other viewpoints.

CONCLUSIONS

Our analytical studies on the reflex contraction of the tensor tympani muscle may be summarized as follows:

1) The latency in this reflex was measured as follows:

Burst of action current of the muscle	6-8 msec
Contraction of the muscle	7-10 msec
Inward movement of the tympanic membrane	18-19 msec

2) From the behavior of the discharge, the components of this muscle may be divided into two neuromusclar units, tonic and kinetic.

3) The threshold of activity of the tonic unit was 80 phones and that of the kinetic unit was 90 phones.

4) The fluctuation of the tympanic membrane in the retracted position under continuous loud sound, discovered by Lorente de Nó etc., is due to the synchronous discharge of almost all the neuromuscular units of the tensor tympani muscle.

5) Acoustic stimulation of one ear evokes reflex contraction of the tensor tympani muscle of both ears.

6) The significance of the tensor tympani reflex is not clear, but supposedly it protects the inner ear against acoustic injury and accommodates it to acoustic stimulation.

The authors wish to express thanks to Professor Cotoji Satta for suggesting this investigation as well as for his constant guidance during the study. Thanks are due to Professor Yasuji Katsuki and Dr. Toshihiko Tokizane for many valuable suggestions.

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LXX

ON THE ARY-EPIGLOTTIC FOLDS

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The ary-epiglottic folds are familiar landmarks to every laryngologist for he inspects them daily in the performance of indirect laryngoscopy. To him their importance lies chiefly in their usefulness as landmarks and the brief mention given them in the anatomical descriptions of the larynx in popular textbooks on otolaryngology[1, 2] is a measure of the practicing clinician's interest in them. If one is suddenly confronted with a problem involving the ary-epiglottic folds, one finds that a great deal of the desired information is not immediately available. Hence, it would seem advisable to assemble some of our knowledge regarding them.

The earliest indication of the future respiratory system is found in embryos of 3 mm, with 20 somites, as the so-called laryngo-tracheal groove which runs lengthwise in the floor of the gut, just caudal to the pharyngeal pouches.[3] The esophagus develops posterior to the groove or diverticulum. Two longitudinal ridges appear in the ventral surface of the esophagus just below the diverticulum, the edges of which fuse, extend cranially, forming a separated canal anterior to the esophagus, closed off except for an orifice just behind the midline hypobranchial eminence or copula (immediately caudal to the second branchial arch). This opening is the primitive aditus to the larynx, bounded above by the hypobranchial eminence, which gives rise to the epiglottis, and posteriorly by the fused upper extremities of the longitudinal ridges, the arytenoid swellings. The lateral connecting folds become the ary-epiglottic folds.[4]

The aperture of the larynx is a rounded triangular opening, base anterior formed by the epiglottis. From the lateral edge of each side of the epiglottis, folds of mucous membrane, the ary-epiglottic folds, extend backwards, downwards, and inwards to the posterior para-

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median line to their origins over the arytenoid cartilages, and here are separated from each other by the mucous membrane of the interarytenoid space. The mucous membrane of these folds enclose muscular fibres, loose connective tissue, and over the apices of the arytenoid cartilages, small nodules of cartilage, the corniculate cartilages (of santorini) and immediately lateral, the cuneiform cartilages of Wrisberg. The areolar tissue allows tremendous swelling as has been demonstrated experimentally by Turner,[5] Miller,[6] and others and is often seen clinically in acute inflammatory and allergic conditions.

The arterial blood supply is from the superior laryngeal artery, a branch of the superior thyroid, communicating freely with the inferior laryngeal, a branch of the inferior thyroid artery.

The venous drainage of the ary-epiglottic folds is into a plexus in the depth of the laryngo-pharyngeal recess (pyriform sinus) from which it is taken by the superior laryngeal vein which penetrates the thyrohyoid membrane. Above, this plexus communicates with the dorsal lingual veins, which extend from the circumvallate papillae to the anterior surface of the epiglottis and the pharyngo-epiglottic fold. Below it communicates with the pharyngeal laryngeal plexus and also anastomoses by two branches with other laryngeal veins; by one branch, with the thyrocricoid vein, and by another with a plexus behind the plate of the cricoid cartilage, which then empties into the inferior laryngeal vein at the crico-thyroid articulation. There are communications between the superior laryngeal vein with the plexus in the membrane of the pharynx over the plate of the cricoid cartilage. There is then, a ring-like plexus lying in the valleculae and along the ary-epiglottic folds, around the entrance to the larynx[7, 8, 9] which may be involved in acute pharyngeal infections. Thus is explained the edema of the ary-epiglottic folds seen in acute inflammations at the base of the tongue such as lingual tonsillitis (occasionally) and epiglottitis (frequently).

The lymph-capillary network is well developed over the mucosa of the ary-epiglottic folds and is drained by the supra-glottic collecting trunks. These, four to six in number, unite near the anterior part of the ary-epiglottic folds, penetrate beneath the mucosa of the pyriform sinuses where they join the lymphatic trunks of the inferior part of the pharynx. They then traverse the thyro-hyoid membrane with the superior laryngeal blood vessels and proceed between the thyro-hyoid membrane and the thyro-hyoid muscles, one toward the greater cornu of the hyoid bone, passing medial to the hypoglossal nerve to terminate in a sub-digastric node: one of several others pass transversely to reach nodes in the vicinity of, or lateral to, the internal

jugular. Still others proceed downwards and outwards and reach nodes placed along the jugular near the omohyoid muscle.[10]

The nerve supply to the ary-epiglottic folds is, in common with the rest of the larynx, through th evagus nerve which receives rootlets from the accessory while passing through the jugular foramen. The superior laryngeal nerve arises from the middle of the modose ganglion, medial to which lies the superior cervical ganglion which gives off rami communicantes to the superior laryngeal nerve.[11] This nerve descends median to the carotid toward the thyroid cartilage and divides into two unequal parts, a large internal and a smaller external branch. The external branch passes downwards upon the inferior constrictor muscle of the pharynx, supplying that muscle and ends in the crico-thyroid muscle. The internal branch of the superior laryngeal nerve pierces the thyro-hyoid membrane and divides into two main branches. The upper supplies the mucous membrane of the lower part of the pharynx, epiglottis, vallecula and vestibule of the larynx; the lower passes medial to the pyriform fossa beneath the mucous membrane and supplies the ary-epiglottic fold and the mucous membrane of the posterior part of the rima glottidis.[12]

In other words, it has been the usual teaching that the internal branch of the superior laryngeal nerve is entirely sensory, though various writers have indicated that the interarytenoid muscle is supplied by the internal laryngeal nerve,[13, 14] or by both the recurrent and the internal branch of the superior laryngeal nerve.[15, 16, 43] More recently it has been quite definitely established that the latter is the case.[17]

The recurrent nerves follow their well known course from below in the groove between the trachea and esophagus, and after entering the larynx supply, with the exception of the crico-thyroid, all of the muscles of the larynx,[11] sending tiny branches, the nervi thyro-epiglottici and ary-epiglottici to the muscles of the same name.[18] At a variable distance before entering the larynx the recurrent nerve divides into two branches, a posterior to the abductor group of muscles, and an anterior branch to the adductors.[19]

The muscular content of the ary-epiglottic folds, Luschka (1871) termed the constrictor vestibuli,[20] there are two recognized bundles. The first, from their origin from the muscular process of the arytenoid cartilage cross obliquely behind the arytenoid transversus muscle and go up and around the corniculate cartilage of the opposite side, to which they are connected by loose connective tissue. All of these muscle fibres do not have a common insertion: some, as a rule the stronger ones, run forwards and upwards in the marginal area of the

ary-epiglottic fold and are connected by very fine tendons to the elastic tissue at the side of the epiglottis: other fibres radiate and end in the quadrangular membrane of the ary-epiglottic fold, and others extend to the infrahyoid portion of the epiglottic cartilage. The second extends from the thyroid cartilage to the epiglottis and may be considered as an extension of the thyro-arytenoid muscle.

Although Luschka states that the muscle fibres of the constrictor vestibuli which extend to the lateral margins of the pars infrahyoidea of the epiglottis are able to produce a necessary infolding of the margins in order to produce many phonatory movements, it is evident from laryngoscopic observations that if the ary-epiglottic folds have any part in phonation, it is a very minor role, and their chief function is inseparably connected with the epiglottis during deglutition.

One understands by deglutition, the passage of solid or fluid matter from the mouth to the stomach, and though very simple in appearance, it is perhaps the most complicated of all the muscular actions connected with digestion, and it is impossible to observe directly. All of the muscles of the tongue, palate and larynx take part.

According to Eykman,[21] the study of swallowing interested the ancients: Hippocrates was investigating the phenomenon by vivisection four centuries before Christ; Gellius (150 A.D.) thought the epiglottis protected the larynx, and Gallen was of the opinion that the food pushed the epiglottis down; Fabricius as Aquapendenta (1537-1619) believed that it was improbable that the epiglottis was pressed down by food, inasmuch as a bit of mucous could then be as effective as a large bolus of food.

Magendie[22] divided the act of swallowing into three stages: (1) the passage of food from the mouth to the throat, (2) the passage over the glottis, and (3) the passage through the esophagus into the stomach. Of the first stage he says, the movements are voluntary with the exception of those of the soft palate. The phenomena here takes place successively and with little promptitude; they are small in number and easily enumerated. But "it is not the same in the second stage. There, the phenomena are simultaneous and multiplied and are produced with such promptitude that Boerhaave considered them as a sort of a convulsion."

Magendie believed the position of the epiglottis was an obstacle against the entrance of food into the larynx but that it was only accessory and the larynx was protected mainly by the movements that close the glottis, and cited two cases of individuals who lacked the epiglottis in which swallowing took place without any difficulty.

Reid[23] in agreement with Magendie, stated that the closing muscles of the glottis can prevent the entrance of food into the larynx after removal of the epiglottis. On the other hand, the epiglottis can prevent ingress of food into the larynx when the movements of all the muscles which diminish the size of the glottis have been suspended by section of all four laryngeal nerves.

Meyer[24] asserted that the larynx is covered by the epiglottis being depressed mechanically by the alimentary bolus, but Czermak[25] (1861) gave a more detailed account of the mechanism of the closure of the larynx, as follows: (1) The arytenoid cartilages intimately meet at their internal surfaces and processes, and they bring the edges of the vocal cords in contact. (2) The superior vocal cords approach the inferior vocal cords so as to obliterate the ventricles of Morgagni; at the same time they also meet in the midline. (3) The epiglottis being lowered and its cushion becomes more prominent still, it presses against the closed glottis; the contact takes place from before backwards. The epiglottis is not passively depressed by the base of the tongue, but by the proper muscles of the epiglottis themselves. He credits Santorini[26] as the first to study the myology of the larynx with correctness and attributing, a priori, to these muscles the effects of lowering the epiglottis.

In 1892 Stuart and McCormick[27] published a paper in which they concluded that the epiglottis never folds down like a lid to cover the entrance to the larynx. This was based on their observation on a 43 year old man who had a fistulous opening in the neck following a number of operations for epithelioma. The internal jugular vein, external carotid artery, contents of the submaxillary triangle, posterior belly of the digastric, the twelfth nerve, a large portion of the constrictor muscles, the greater part of the greater cornu of the hyoid bone, part of the parotid gland, faucial pillars and tonsil, internal carotid, and one inch of the common ramus of the jaw had been sacrificed. They had attempted to observe the swallowing of food through a glass plate placed over the external opening but the food adhering to the glass obscured vision so they looked directly into the throat while the subject swallowed oysters. These passed over the epiglottis, which never "folded back." In eating, the man placed his hand on the pad (closing the fistula) and then "swallowed quite well." But in swallowing liquids, sometimes they "got into the wrong throat." This was explained by (1) imperfectly contractile wall of the channels, (2) one side of the lower jaw being absent, the elevators of the hyoid were not efficient and (3) the hypoglossal and most of the facial nerve of one side were wanting.

In denying the closure of the larynx by the lid-like action of the epiglottis, Stuart[28, 29] (1891-92), explains the mode in which food is prevented from entering the larynx. Closure of the glottis, as in forced action, is not sufficient, for if the superior aperture were left open it would constitute a funnel to take in particles of food, which would require violent expiration to expel. Otherwise they would fall of their own accord, or be inspired, into the lower air passages.

When the entrance of the larynx is to be closed, said he, the arytenoids leave the posterior wall of the pharynx, are rotated and moved bodily inwards and forwards, and the upper parts with the santorinian cartilages fit into the grooves formed by the incurved margins of the epiglottis base. The muscles concerned are mainly the external thyro-arytenoids. The ary-epiglottic muscles, including the oblique arytenoids, tense the ary-epiglottic folds. The elevators of the larynx pull it upwards to engage against the base of the tongue with the base of the epiglottis compressed between. In addition to tensing the ary-epiglottic folds the ary-epiglottic muscle is supposed to bend the lateral edges of the epiglottis backward, and assist the thyro-arytenoid muscle in rotating inwards and pulling forwards the arytenoids.

Kanthack[30] criticized stuart's work and stated that although the exact function of the epiglottis was not fully understood, that in animals such as Stuart used in his experiments the epiglottis did act as a laryngeal lid when swallowing in the normal position.

Renshaw[31] lists as one of the objections to the theory that the epiglottis acts as a lid over the larynx during deglutition, that the muscles attached to the epiglottis, consisting of a few fibres of the aryteno-epiglottideus and part of the thyro-arytenoideus externus are weak and incapable of bending the cartilage transversely as would be necessary for them to do in order to depress it over the larynx. Their function seemed to him therefore to draw back the two lateral edges and to make the ary-epiglottic folds tense.

He attempted to observe the act of swallowing with a laryngeal mirror. Although obviously unable to observe the complete action of the epiglottis and larynx, he reported a backward movement of the epiglottis, during which the lateral margins are drawn backward and downwards by the ary-epiglottidean muscles thus making the posterior surface of the epiglottis more concave from side to side. Still, he concludes that the chief function of the epiglottis (and ary-epiglottic folds) is to deflect saliva and secretions from the nose and nasopharynx into the pyriform sinuses.

Eykman[21] was a pioneer in the study of deglutition by means of the x-ray. With his early equipment an apparatus was arranged so that the upward movement of the larynx induced the current to the x-ray tube and this had to be repeated again and again in order to produce an image on a photographic plate. From his experiments he concluded that during deglutition the larynx is protected by closure of the glottis, the arytenoids being brought forward, the inferior half of the epiglottis (laryngeal part) closes in the space between the false cords and the arytenoids. The closure by the epiglottis is affected in three ways, viz., pressure by the base of the tongue, pressure backward by the sub-hyoid fat pad which is displaced posteriorly in the elevation of the larynx as the superior border of the thyroid cartilage comes in contact with the hyoid bone, and by a "swallowing inhalation" which aids in drawing the epiglottis tightly into the superior aperture of the larynx.

There have been some bizarre descriptions of the act of swallowing, in which for example the epiglottis is credited with closing the larynx by being pressed down over it, but with also the tip slipping downward along the back wall of the pharynx, "pushing the bolus, probably with a final quick thrust, into the gullet!"[32]

Mosher[33] after studying the act of swallowing by means of x-ray, was emphatic in stating the epiglottis does actually turn downward to cover the larynx, the cushion of the epiglottis approximating the tips of the arytenoids. This closure is made complete by the sphincter-like action of the muscle fibres in the ary-epiglottic folds. He states that although the upper half of the epiglottis can be removed or destroyed and difficulty does not result, but if the base or cushion of the epiglottis is interfered with, difficulty in swallowing does result.

At almost the same time Negus[34] was insisting that the theory of the epiglottis acting as a lid over the larynx to prevent the ingress of food or water during swallowing was erroneous.

Barclay[35] gave an account of normal swallowing in which he maintained that the bolus is carried from the back of the tongue to the clavicular level largely by negative pressure. The mechanism producing the negative pressure is simply the sudden opening of the esophagus with all other exits (mouth, nasopharynx, larynx) tightly closed. The opening into the larynx is cut off by a dragging up of the laryngeal pharynx behind the larynx to form contact with the epiglottis.

Ducuing and Ducuing[36] likewise in a lengthy article on swallowing concluded that the protection of the larynx was less depend-

ent upon the lowering of the epiglottis than it is on the elevation of the laryngeal turbinal against the base of the tongue.

Pancoast[37] held that the epiglottis and not the back of the tongue closes off the air passage. With this Barclay[38] agreed but insisted that it is the back of the tongue that pushes the epiglottis backward while the larynx is raised to meet it, and hence the epiglottis takes no vital part in closing off the larynx in swallowing. He contended like Stuart that the epiglottis remained vertical and "stands out like a rock under a waterfall!" Ordinarily food supposedly goes over without touching it, but some of the thick sticky food he used seemed to divide on it and be caught in the valleculae or be diverted to both sides, passing round and down into the esophagus in one or both the ridge channels formed by the ary-epiglottic folds. He observed in his roentgenograms that the upper part of the larynx—the vestibule right down to the false cords—is obliterated during the closed phase of swallowing. The hyoid and thyroid cartilage rise together, and "something comes from above and slips down between the hyoid and the epiglottis forming a well rounded shadow over the top of the larynx and obliterating the whole laryngeal vestibule." This he was unable to explain.

On the other hand Welin[39] states that radiographically it was easily observed that the epiglottis does bend downwards from its upright position.

Johnstone[40] also demonstrated by radiography that the epiglottis does actually turn over. He explains this movement as resulting from interacting forces: (1) the larynx is drawn up first by the hyoid bone which also moves forwards, and then by a marked elevation of the thyroid cartilage, causing the pad of fat at the base of the epiglottis to bulge backwards pushing out the tubercle of the epiglottis so that it readily meets the closed arytenoids as they move upwards and forwards, (2) the backward push of the tongue and (3) the contraction of the small epiglottic muscles. Furthermore, if several swallows are made without interruption for respiration, then the epiglottis remains overturned until the act is completed when the hyoid bone and thyroid cartilage drop back and the base of the epiglottis pivots round so that the tip is a little bent over as it slides up the posterior pharyngeal wall. The restoration is completed by the recoil of the elastic tissue. Johnstone was once fortunate enough to see the epiglottis turn over in a patient with a lateral pharyngostomy—an observation in direct opposition to that made by Stuart on a similar case.

Johnstone is critical of Barclay's reports, saying that the negative pressure in the pharynx and upper esophagus cannot be confirmed.

He is also certain the something that "comes from above and slips down between the hyoid and the epiglottis, forming a well rounded shadow across the top of the larynx and obliterating the whole larynx down to the false cords" which Barclay is unable to identify, is the epiglottis itself. He admits that after removal of the epiglottis there is no difficulty in swallowing, and this is used as an argument in favor of the upright theory, but it should be remembered that in surgical removal the base of the epiglottis is not excised.

Negus[41] reiterated that the epiglottis did not fold back like a lid over the larynx during swallowing and that its influence on deglutition is slight, being merely pressed against the base of the tongue, and aiding, as the cartilage of Wrisberg and the arytenoids are pulled forwards against its cushion. The base however gives support to the ary-epiglottic folds, and thereby assists in forming a lateral food channel. The function of these folds are connected entirely with deglutition.[42] When liquids are trickling past the larynx in small amount, they are prevented from entering the glottis by these lateral barriers. If the glottis be open the backward pull of the posticus muscles braces back the ary-epiglottic folds and stretches them, so that with the aid of the cartilages of wrisberg they stand up above the level of the pharyngeal floor, leaving on either side a food channel. When a big bolus has to be swallowed the postici and cricothyroid muscles are relaxed and the ary-epiglottic folds are pressed against each other by sphincteric action of the thyro-arytenoid and inter-arytenoid muscles, and particularly by those bundles known as the thyro-epiglottic and ary-epiglottic muscles. Thus it is by the closure of these folds that the larynx is protected during deglutition.

Pressman[43] has described the sphincter-like action of the superior aperture of the larynx. When called into play (in swallowing) the muscle fibres in the ary-epiglottic folds contract, approximating these structures one to the other, and pulling the epiglottis downwards so that the tubercle of the latter is in contact with the puckered sphincter, while the posterior gap is filled by the arytenoid mass.

In a later communication Negus[44] retreats somewhat from his former position and concedes that although the epiglottis may fold over the aperture of the larynx, it does so as a result or other movements designed to close the airway, and without any active role of importance. At the same time he stresses the closure of the larynx by the sphincteric girdle of muscles in the ary-epiglottic folds. By their action the antero-posterior diameter is shortened, and the lateral margins are pressed together and against the cushion of the epiglottis.

Thus it is seen that although the mechanism involved in the closure of the larynx during deglutition has been the center of great

interest, the opinions regarding it have varied considerably. This is understandably so inasmuch as the actions involved are impossible to observe directly, and the complexity and rapidity with which they occur make them likewise almost impossible to analyze accurately when observed fluoroscopically. Even the views held by competent radiologists were contradictory because of the difficulty in securing films at exactly the desired instant.

Settlement of the controversial question was to await the development of cine-radiography. Yet Frenckner[45] (1948) while able to record the movements of the epiglottis fairly well at a speed of sixteen exposures per second, was unable to prove to his own satisfaction that the epiglottis was or was not necessary to prevent the food trickling down into the larynx.

Recently it has been demonstrated that relatively high speed cine-radiography is necessary to analyze the second stage of swallowing. At the University of California, Saunders, Davis and Miller[46] were able to photograph the swallowing sequence at speeds of thirty and sixty frames per second and establish without doubt that the epiglottis does act as a trapdoor sealing the entrance to the larynx during deglutition. Indeed the epiglottis was observed by them to bend backward to thirty and even forty-five degrees below the horizontal so that the subject seemed "to have swallowed his epiglottis." The final tipping of the epiglottis they found, occurs with such extraordinary rapidity that Frenckner was unable to find absolute evidence of it since these movements often occur in less than 1/15 second. These investigators were also able to show definite shortening of the ary-epiglottis folds so that they feel that the contained muscles may assist in pulling the epiglottis down and back, although its descent is likely controlled by both active and passive factors. They are further of the opinion that the constrictor mechanism is so efficient that of itself the larynx could be protected from entrance of ingested food, as is said to occur in cases of excision of the epiglottis.

Their work is of the greatest value and has settled definitely the question over which there has been the most controversy, i.e., the behavior of the epiglottis. One may differ with them in their conclusions regarding the modus operandi. The epiglottis may be regarded as a trap-door, counter balanced by its ligaments much like an over-head garage door. Its backward excursion begins with the elevation of the larynx and the backward movement of the tongue and it is only this beginning movement that is passive. The actual closure of the larynx, like all involuntary motions, is active and brought about by the intrinsic muscles, chiefly by the muscles of the ary-

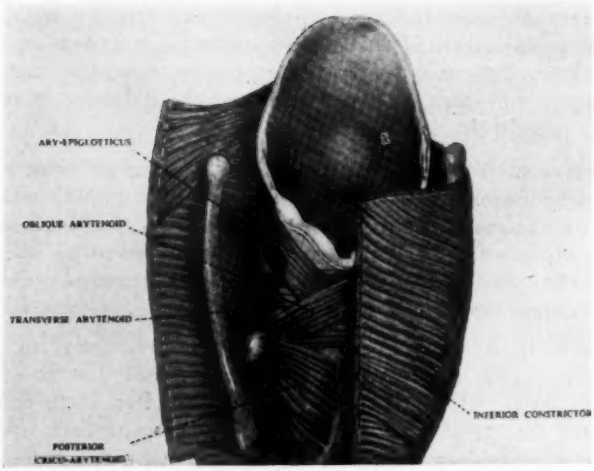

Fig. 1.—Showing muscles in the ary-epiglottic folds (from Scott-Brown). Courtesy: Butterworth and Company, Ltd., London.

epiglottic folds. The arytenoids are brought together by the adductors (inter-arytenoids, lateral crico-arytenoids, thyro-arytenoids). Contraction of the fibres of the ary-epiglotticus that end in the quadrangular membrane serve to tense the lower portion of the ary-epiglottic folds; those in the marginal portion slide the corniculate cartilages forward and shorten the A-P diameter of the larynx, and by traction on the epiglottis bring it back so that the tubercle closes over the puckered opening. The thyro-epiglotticus adds its downward pull which further seals the closure of the epiglottis over the tensed ary-epiglottic folds. If there is an almost insensible contraction of the diaphragm at this instance (the swallowing inhalation of Eykman), it would serve to seal the closure the more perfectly. Each added safeguard is of importance for the pressures developed in the pharynx are almost unbelieveable. Thus Falk and Kronecker[47] found that during the initiation of the act of swallowing the closed buccal cavity showed a manometric pressure of 20 cm of water; Negus[44] mentions that in one patient with a pharyngostome a pressure of 100-200 cm of water was recorded, while Allison[48] demonstrated that by the force of swallowing alone a column of water two and one-half feet could be maintained!

That the ary-epiglottic folds are of utmost importance in the active closure of the larynx can be observed in clinical cases, any number of which could be enumerated. The following three given in brief are typical:

F. R., male, aged 75 years, was seen April 18, 1951, complaining of sore throat of two days' duration. In the past 24 hours hoarseness appeared and swallowing was quite painful. He was so miserable that he was unable to sleep the night before. On this date he was very apprehensive and choked on taking fluids. His temperature was only 99.6, pulse 100. There was marked edema of the arytenoids and ary-epiglottic folds, redness but no swelling of the epiglottis and false cords. There was no actual dyspnea but because of the rather severe edema he was hospitalized until all swelling had subsided, a matter of a few days.

D. L., an elderly female was seen March 21, 1952. At three o'clock in the morning she had risen and taken an aspirin tablet which seemed to "stick in her throat." The sensation persisted; early in the day she felt like she was choking, and later in the day strangled on attempting to swallow the smallest sips of water. The temperature was normal, but she was very anxious and apprehensive and was afraid to lie down. The only finding was edema of both ary-epiglottic folds, extending on to the arytenoids. The epiglottis, true and false cords were unaffected. She was treated symptomatically. In 24 hours there was much improvement and in 48 hours complete recovery.

Mrs. R. V. S., a 43 year old nurse was seen May 31, 1952, in consultation. She had been well the day before and not until retiring did her throat begin to "feel funny." On waking there was pain on swallowing and she choked and strangled on taking even sips of fluid. She was extremely apprehensive, and insisting on sitting upright, being fearful of lying down. The temperature was 100.6, the pharynx was normal, but mirror examination showed redness of the epiglottis, and the mucosa of both ary-epiglottic folds was edematous. The strangling persisted for 24 hours and there was some difficulty in swallowing for the next 24 hours. Thereafter swallowing was normal, the edema of the ary-epiglottic folds having subsided.

Common to these cases, and in all others in which there is edema of the ary-epiglottic folds, is a fearfulness and apprehension which is apparently due to the choking and strangling on attempting to swallow even the smallest amounts of fluids. When inflammation is limited to the epiglottis alone there is pain on swallowing, so that

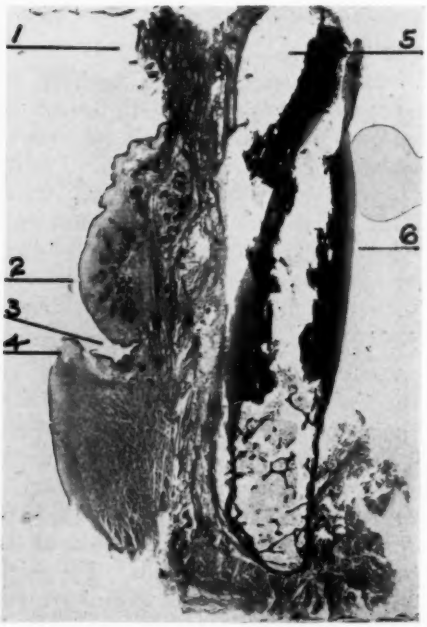

Fig. 2.—Cross section through the wall of the larynx showing well developed musculature in the ary-epiglottic folds. 1, ary-epiglottic fold; 2, false cord; 3, ventricle; 4, vocal cord; 5, pyriform sinus; 6, ala thyroid cartilage.

a child may drool and even refuse to swallow, but there is no strangling unless the ary-epiglottic folds are involved. Since there is no limitation of elevation of the larynx, or of the movements of the tongue, there is no impediment to the factors that influence the passive motion of the epiglottis. Now if the entrance of the larynx were sealed by the passive lowering of the epiglottis, the edema of the lateral margins of the aperture would serve to make the closure more secure. That the opposite is true is certain because of the strangling on swallowing, for liquids obviously "go down the wrong throat." It must be therefore that the edema interferes with the contraction of the muscular content of the ary-epiglottic folds so that the edges are unable to tense adequately and their power to lower the epiglottis completely, is lost. In these cases, glottic closure alone, which is unimpaired, is insufficient to prevent fluids from

entering the airway. It argues in favor of the statement that if the cushion of the epiglottis is removed, difficulty in swallowing will ensue. In fact it is the epiglottis that keeps the anterior attachments of the ary-epiglottic folds apart. If the infrahyoid portion of the epiglottis were removed, the anterior edges of the ary-epiglottic folds might be drawn together causing respiratory difficulty (Fig. 3).

There has been much speculation as to the reasons for the different behavior of the vocal cords in partial or complete recurrent nerve paralysis while at the same time little thought has been given to the fact that the sphincter of the entrance to the larynx continues to function.

As is well known, in cases of bilateral abductor paralysis resulting from thyroid surgery the onset is sudden and in addition to dyspnea there is a choking and strangling on taking liquids but the latter symptom disappears in a few days. It is unlikely that it is because the posterior (abductor) trunk is multi-branched, as Trustad and Morrison[49, 50] have shown may occur, allowing the nerve fibres to the postici muscles in one branch to be interrupted while the filaments to the ary-epiglottic muscles in another branch are spared. King and Gregg[19] ascribe the nerve supply to the latter muscles to the posterior or abductor branch, but admit that this may be in error inasmuch as they are part of the constrictor or defense mechanism of the larynx, and since the anterior branch supplies the constrictor group of muscles, it may also supply the ary-epiglottic muscle. This explanation is particularly applicable to those patients who have had a King operation for the relief of dyspnea caused by bilateral abductor paralysis. In a successful operation of this type the patient is unable to close the glottis and must depend on the integrity of the constrictor mechanism of the superior aperture, the ary-epiglottic muscles, to protect the larynx during swallowing.

This explanation does not apply to complete (abductor and adductor) paralysis of the recurrent nerve. If this is of sudden onset, there is choking and strangling which disappears in a few days, so that the superior sphincter remains intact. We can agree with Pressman[43] that in these cases, though there is no movement whatsoever of the affected true cord, movement can be detected in the region of the false cord. Since it is now established without doubt that the inter-arytenoid muscle, a part of the constrictor mechanism, is supplied by the internal branch of the superior laryngeal nerve (as well as by the recurrent), it is quite possible the aryepiglottic muscle, the constrictor vestibuli, also receives motor fibres from the internal branch of the superior laryngeal.

Fig. 3.—The ary-epiglottic folds attached to the dorsum of the epiglottis meeting in the midline causing supraglottic laryngeal obstruction. Both false and true vocal cords are obscured. Note that the prominence of the corniculate cartilage and the recess of the pyriform sinuses are obliterated.

As has been stated, the ary-epiglottic folds are capable of tremendous swelling, and yet it is seldom that such swelling produces laryngeal obstruction: if it occurs it is likely due to an allergic state. Laryngeal obstruction in acute infectious conditions is more apt to be due to intense inflammation and swelling of the false cords.[51] Chronic laryngeal obstruction by the ary-epiglottic folds is even more unusual, but it may occur as is illustrated by the following case:

R. E. MacN., aged 26 years, was brought to the U. S. V. A. hospital on October 6, 1947 after having ingested poison, later found to have been cuprous chloride and mercury chloride, amounts undetermined. There was considerable swelling of the mouth and throat and he was watched closely for indications demanding tracheotomy. The swelling subsided however without the need of tracheotomy. He developed a nephrosis and gastrointestinal symptoms which cleared up. Because of hoarseness and some difficulty in swallowing he was bronchoscoped and it was reported that there was swelling of the supraglottic region and some edema of the vocal cords, but no obstruction. The esophagus was studied with barium and no constriction found, and he was discharged on November 22, 1947.

He was re-admitted July 5, 1949, with the complaints of shortness of breath and hoarseness. Since leaving the hospital the shortness

of breath had been increasing and there was a loud wheezing on any kind of exertion. The sound seemed to originate in his throat.

Physical examination was negative except for the laryngeal examination. This was described as a "stricturing of the lateral laryngeal walls" in the supraglottic region, with considerable redundant mucosa which appeared to be edematous and swollen in the region of the ary-epiglottic folds. The redundant mucosa was described as falling over the vocal cords, restricting the airway, thus causing the hoarseness and dyspnea. Laboratory findings including urinalysis, blood count, serology, total protein, b. u. n., etc. were normal. Chest x-ray was normal.

On July 12, 1949, a tracheotomy was performed, and over the next few months a number of direct laryngoscopic examinations were done. Biopsy of tissue from the ary-epiglottic folds showed only inflammatory reaction. Dilatations were carried out without benefit of any kind. Attempts to incise the folds with a knife were unsuccessful, the tissue acting like an elastic band moving away from the knife edge. Even x-ray therapy was resorted to, 1650 r to each side, without benefit.

It was then that the patient came under my observation. Mirror laryngoscopy showed the anterior ends of the ary-epiglottic folds attached not to the lateral edges of the epiglottis but to its posterior surface clear across to the midline, but not to one another. The attachment seemed to be on a plane higher than usual and the A-P diameter of the larynx seemed shortened. The space between the folds was a mere slit so that neither the false nor the true cords could be seen. There was no prominence over the arytenoid cartilages, and the drawing of the ary-epiglottic folds toward the midline obliterated the recesses of the pyriform sinuses giving the appearance of a flat horizontal floor to the hypopharynx (Fig. 3).

It was clear that the abnormal attachment to the epiglottis must be severed, and re-attachment prevented. That scarring might shorten their length and result in some stenosis in the A-P diameter was considered.

At direct laryngoscopy attempts were made to sever the attachment to the posterior surface of the epiglottis with a knife only to find, as had the previous operator, that the tissue simply stretched away from the knife, and with sawing motions it also moved with the knife.

It was decided then to utilize the cutting current by indirect vision with an electrode fashioned like the cautery tip advocated by

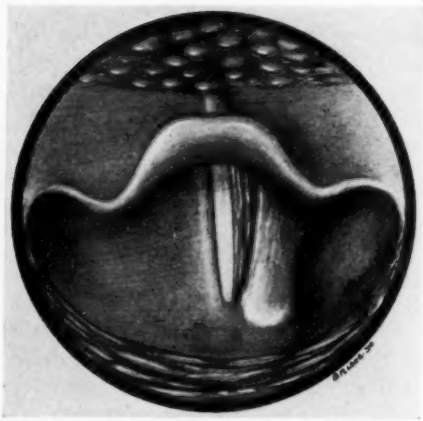

Fig. 4.—Mirror image following operation on the left ary-epiglottic fold. The left false and true vocal cords can be seen on phonation. Note the return of the prominence over the arytenoid and restoration of the recess of the pyriform sinus.

G. B. Wood[52] for ignipuncture of tubercles of the larynx. This was done very gingerly to the left side on December 21, 1949, and then observed for some time to see if scarring might shorten the length of the fold. None was noted after several weeks. The attachment to the posterior surface of the epiglottis had been shifted over on this side from the middle to halfway toward the lateral edge, so that the vocal cord of the same side could be seen (Fig. 4).

On March 6, 1950, the opposite side was attacked in the same manner with more confidence. This side healed without scarring, and the attachment was moved over to its normal site at the lateral edge of the epiglottis. The entire length of the false and true cords could be seen, the area of prominence returned over the arytenoid cartilages and the pyriform sinuses regained their depth. Now with the tracheotomy tube corked he could run up and down stairs without any dyspnea. He was decannulated and discharged June 9, 1950.

SUMMARY

Because the usual descriptions of the ary-epiglottic folds are so brief as to be almost valueless to the clinician, the anatomical facts having a clinical application are brought together.

The function of the ary-epiglottic folds is discussed. In the literature on the second stage of swallowing, the role of the epiglottis has excited the most interest. It is pointed out that the ary-epiglottic folds are of equal importance in the protection of the airway during swallowing.

From clinical observations it would appear that the sphincters of the superior aperture of the larynx derive a greater portion of their motor nerve supply from the superior laryngeal nerve than has been demonstrated anatomically.

It is usually considered that the ary-epiglottic folds may cause laryngeal obstruction only in acute conditions. A case of chronic laryngeal obstruction caused by the ary-epiglottic folds is described.

9 Exchange Place.

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LXXI

CARCINOMA OF THE VALLECULA

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Newark, N. J.

The backward-lying epiglottis has long attracted its share of attention for obscuring the anterior part of the larynx where growths may reside for a considerable time before discovery. I wish to concern myself in this paper with a less discussed subject, that of carcinoma of the vallecula, evading detection because of a *forward*-lying epilottis. Unlike cancer arisin on the posterior surface of this structure, it may give rise to early and rather characteristic symptoms.

Anyone who has removed a foreign body from the vallecula or who has himself experienced the discomfort of a foreign substance lodged in this site is well aware of its sensitivity. It is my belief that early cancer in the vallecula produces symptoms which bring the patient to the physician for relief at a time when this disease can be treated with excellent possibilities for cure, provided the examiner looks with sufficient care to detect it.

Every otolaryngologist is aware of the variation in shape, size, and mobility of the epiglottis. Some are flat, broad and stiff. Others tend to varying degrees of curvature, culminating in the characteristic omega-shaped epiglottis so often described in the congenital laryngeal stridor of infancy and often persisting into adult life. Still others lie against the base of the tongue in normal quiet respiration.

It is this latter type which chiefly concerns us, since it may easily obscure the vallecular sulci. Three factors militate against adequate inspection of this area: First is our natural eagerness to see the vocal cords. Second is their being "roofed over" by the tip. In some cases, phonation, with a high "E-e," will erect the epiglottis to such a degree that the valleculae become visible. But in others it will not. Third, persistent hypertrophy of the lingual lymphoid tissue, either by itself or in combination with a forward-lying or curved epilottic tip, may interfere.

342 cases have been observed. In 64 cases, or 18.7% the valleculae could not be seen. The only way in which adequate visualization could be obtained was by backward displacement of the tip of the epiglottis with a curved laryngeal syringe under local anesthesia.

There is the most amazing variety in the conformation of the structures at the base of the tongue; in depth of the sulci, in the prominence or the absence of the median bar, in the shape of the space between the epiglottis and tongue on phonation and on quiet respiration and in the presence or absence of "occult" valleculae.

REPORT OF CASES

CASE 1.—W. V., a 58 year old white male, was first seen October 21, 1951 complaining of sore throat. For about six or seven weeks he had been conscious of a soreness in the left side which was unremitting but which did not increase in severity. He had been treated without effect by his family physician and complained of pain on swallowing certain liquids such as fruit or vegetable juices. His past history contributed nothing of interest.

Physical examination. There was a red, inflamed, lateral lymphoid band running down the posterior pharyngeal wall on the left side from just above the uvula to the level of the base of the tongue. Cords, arytenoids, pyriform sinuses and valleculae were normal and there were no enlarged cervical nodes. Culture from the pharynx revealed a pure strain of hemolytic streptococcus; the blood-count was essentially normal. The patient tried a local antibiotic but in a week said that he felt about the same. Biopsy of the inflamed lymphoid band on the left posterior wall showed only chronic inflammatory changes.

The patient was kept on various types of local treatment for two months without appreciable improvement and was finally admitted to St. Barnabas Hospital on December 12, 1951 for excision of the lymphoid band. He was discharged the following day and pharynx healed in ten days. There was temporary improvement in his symptoms but he had recurrence of the soreness lower down by the end of December. In January he was given a course of parenteral penicillin but this did not help. In February he was referred to another otolaryngologist who admitted him to another hospital and biopsied the left pyriform sinus. The biopsy showed hyperkeratosis but no other lesion. He had a thorough examination at this time including a barium swallow and esophagoscopy. The patient was referred back to me with symptoms still persisting and on repeated examination there was no adequate explanation for the complaint. Finally five months after the initial examination a lesion was found in the base of the left vallecula on the anterior surface of the epiglottis by using a laryngeal syringe to move the latter away from the tongue. A specimen was taken and showed anaplastic epidermoid carcinoma. There was no cervical lymphadenopathy.

Looking backward over the record at this point one wonders why the patient continued to submit himself for repeated examinations which disclosed nothing. A source of self-recrimination has been the note made on my initial examination that the valleculae showed no pathology. This has cautioned me against the casual insertion of such an observation in a patient's record unles the site in question has been thoroughly scrutinized.

The patient was re-admitted to hospital.

Physical examination. Showed a well developed, somewhat obese white male apparently in good health. The lesion in the left vallecula measured about 1.5 cm as described. There were no enlarged cervical nodes. The heart sounds were regular with no murmurs or enlargement and the blood pressure was 120/80. The lungs were clear to percussion and auscultation, the abdomen was obese but there were no abnormal masses. Reflexes were normal.

Laboratory examination. Urinalysis and blood count were normal. X-ray films of the lungs showed no evidence of metastatic disease, but there were small areas of fibrosis in the right lower lobe.

On March 20, 1952 an anterior transverse pharyngotomy was done under pentothal and nitrous oxide anesthesia. An endotracheal tube was used up to the point at which the pharynx was entered, when a tracheotomy tube was inserted through a previously prepared incision in the suprasternal notch. The patient received 500 cc of blood during the operation.

Postoperatively nutrition was maintained with a nasal feeding tube. The temperature fluctuated between 100° and 102° for the first few days gradually subsiding to normal in one week. The patient received penicillin 400,000 units twice daily. The sutures were removed on the fourth postoperative day, the tracheostomy tube was corked on the seventh and the feeding tube and tracheostomy tube were removed on the eighth. The patient was able to swallow at that time, although with an occasional cough. He was discharged on the fourteenth postoperative day with the wound healed and in good condition.

He has been followed at monthly intervals in the office since and has been able to eat a regular diet although he has to be careful when he swallows and has a glass of water at hand to aid in deglutition. There has been no evidence of recurrence two years following the operation.

CASE 2.—J. B., a 74 year old white male, was admitted to St. Barnabas Hospital June 16, 1949 complaining of vague pain in the

right side of his throat on swallowing, of one month's duration. His past history was entirely negative. He was a heavy smoker.

Physical examination. Urinalysis and blood count were normal. A barium swallow showed some difficulty in initiating deglutition but otherwise the esophagus was normal. An esophagoscopy was done the day following admission but nothing was found except for a spur 15 cm from the upper teeth. The patient was discharged on June 21, 1949 to be followed in the clinic.

He was observed there until August 1949 when an ulcerative lesion was noted in the left vallecula and biopsy was advised.

On September 13, 1949 a specimen was taken from the vallecula through a laryngoscope with a resultant hemorrhage which required tracheostomy and pharyngeal packing. There was a febrile course with subcutaneous and mediastinal emphysema and signs of aspiration pneumonia which gradually cleared under antibiotics. The biopsy was capillary hemangioma. The patient was decannulated September 19, 1949 and discharged September 21, 1949.

On October 1, 1949 the patient was again admitted because the ulcerative lesion in the vallecula was still visible. A biopsy done on October 4, 1949 showed epidermoid carcinoma.

The physical examination showed essentially no change since the previous one except for an elevation of blood pressure to 170/90.

Laboratory examination was not significant and x-ray of the chest was negative for metastases. There was 1400 cc deficit in blood volume. Electrocardiogram disclosed occasional premature beats but was otherwise normal.

The patient was adjudged a fair risk for a man of 74, by the medical consultant, and was given a transfusion of 500 cc of blood. On October 17, 1949 an anterior transverse pharyngotomy was done, preceded by a tracheostomy. The epiglottis, the base of the tongue and the hyoid bone were resected, including the tumor.

The postoperative course was fairly smooth with the temperature rising to 101° for one week and gradually subsiding to normal. The patient was kept on antibiotics and a feeding tube was used for the first week. He developed a temporary pharyngeal fistula which healed by the tenth postoperative day. The tracheostomy tube was removed on November 5, 1949. There was considerable coughing while the patient was eating but a barium swallow showed no spillage into the tracheobronchial tree. The patient was discharged on November 21, 1949 with his wound healed.

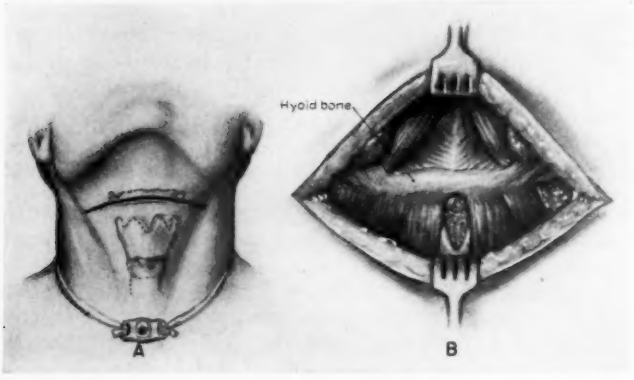

Fig. 1.—Incision for anterior transverse pharyngotomy with exposure of the field preparatory to section of muscles attached to the hyoid.

Since then he has had six hospital admissions for coughing, abdominal pain or difficulty in swallowing. On one occasion he had a definite right lobar pneumonia which responded to chemotherapy. Repeated barium swallows have shown no evidence of obstruction or malignant disease and esophagoscopies on several occasions have been negative. Chronic alcoholism has been a factor.

The region at the base of the tongue has remained free of disease and there have been no cervical metastases. The patient has maintaind his nutrition fairly well although he has continued to complain of cough while eating. He was last examined August 8, 1951 and now lives in a home for the aged.

COMMENT

It will be noted that in the second case there was a delay of two months between the initial visit of the patient to the clinic and the discovery of the lesion in the vallecula, and a delay of another six weeks before a positive biopsy was secured. In the first case there was a similar delay of about five months between the time the patient sought relief and the establishment of the diagnosis. The pain of which these patients complained was consistently referred to one side of the throat. It was aggravated by swallowing and was described as burning in character, especially on ingestion of tart juices, or hot or highly seasoned foods. There is no reason to believe that an incipient tumor with intact surface in the vallecula causes dis-

comfort any more than does a similar lump in any other place such as the lateral pharynx or tonsil. Without doubt the burning pain of which both these patients complained depended upon the presence of ulceration, which must have appeared early, since at the time of excision neither lesion was more than 1.5 cm in its greatest dimension.

Other than this one complaint neither patient manifested any clue to his difficulty. Both maintained their weight and carried on normal activities.

A perusal of the histories of these patients emphasizes the old aphorism that one not only must look, but must see. The physicians who carried out the earlier phases of the workup were conscientous; they conducted the usual routine indirect examination of the larynx; they had x-rays with barium and they did esophagoscopies, but they failed to inspect all of the places into which they might have looked; and in one of the cases I was the first examiner. Only when the tip of the epiglottis is displaced backward under adequate local anesthesia does the site of the disease become apparent. A biopsy is of course essential to confirm the diagnosis.

One is tempted after experiences of this kind to make a checklist of the various sites in the upper respiratory tract where cancer can occur—but then, where does it not occur? I have been driven, instead, to conduct an unremitting search for a primary lesion as long as the patient's symptoms remain unexplained, anesthetizing the mucous membranes when necessary so that no cul-de-sac like an occult vallecula may conceal an early growth.

Well aware that cancers involving the base of the tongue are ordinarily treated by crossed radiation sometimes supplemented by radon seeds, I was at first disposed to refer both of these patients for such treatment. Since the lesions were small it seemed feasible, to excise them and preserve the functions of deglutition, respiration, and speech. If one preserves both hypoglossal and both superior laryngeal nerves, there should ensue no permanent impairment of swallowing, although I have seen patients who have had such difficulty following pharyngotomy.

The choice lay between anterior transverse pharyngotomy and lateral transthyroid pharyngotomy. Trotter devised the latter procedure for growths of the lateral and posterior hypopharyngeal wall as well as for those of the aryepiglottic fold and postcricoid region.[13-15] Orton,[8-10] perhaps more than any other in this country, has advocated and used Trotter's procedure.

For the type of lesion with which we were dealing however, somewhat higher and more centrally placed, the anterior approach offers

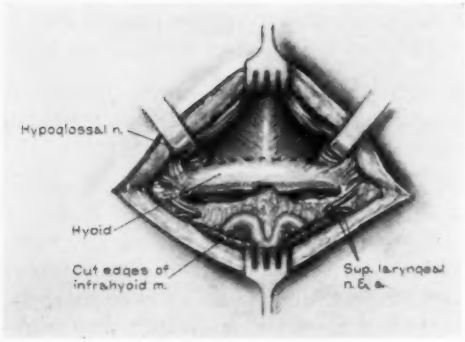

Fig. 2.—The infrahyoid muscles have been sectioned, the digastric freed and the hypoglossal nerves isolated. Incision of the attachment of the mylohyoid is shown by dotted line.

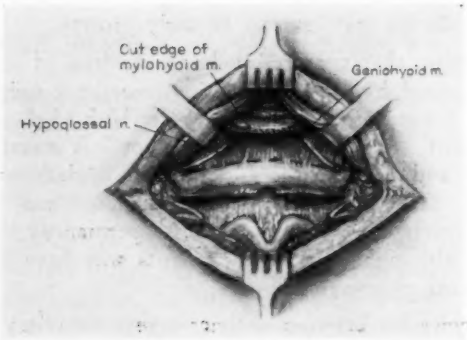

Fig. 3.—Mylohyoid muscle has been sectioned and line of incision through geniohyoid and hyglossus muscles is shown.

certain advantages. The angle of the mandible does not intrude as it does in the lateral approach. The superior laryngeal nerve does not suffer as much damage as it does when retracted downward and backward in a lateral field. Finally closure, especially of the opposite side, is more difficult from the lateral than from the anterior approach.

Portmann,[11] Blassingame,[1] and Klopp and Delaney[6] have all recommended anterior pharyngotomy. Blassingame's recapitulation of the historical background of pharyngotomy will interset those who wish to know more of this subject. There is some disagreement over the point of entry into the pharynx, whether above or below the hyoid bone, and over the disposition of the hyoid itself. If one is dealing with cancer in proximity to the latter, its inclusion in the resected specimen would seem mandatory. The name, "anterior transverse pharyngotomy," is suggested for this type of procedure.

In all head and neck cases during the past eight or ten years pentothal supplemented by nitrous oxide and oxygen delivered through an intratracheal tube has provided the safest anesthetic and afforded the fewest postoperative problems especially in the older age group which comprises the bulk of patients with malignant disease. Both patients received this anesthetic.

It is advisable to prepare the tracheostomy site immediately, even though the trachea is not entered or the tube inserted until a later stage in the operation. For this purpose a short vertical or horizontal incision best fulfills the requirement of isolating this part of the procedure from that which follows. Splitting of the deep fascia and if necessary of the thyroid isthmus completes the preparation for insertion of the tracheostomy tube at any time the operator may choose. This eliminates the delay in shifting from one type of airway to another at some critical point. A transverse incision at the level of the hyoid bone and reaching the sternocleido-mastoid muscles at its extremities is carried down through the platysma and the flaps reflected upward and downward (Fig. 1). Sharp dissection severs the ligamentous attachment of the digastric muscle to the hyoid bone on each side and the muscle retracts upward. This enables one to isolate the hypoglossal nerve which is dissected free and likewise allowed to retract (Fig. 2). The mylohyoid muscle is cut at its attachment to the hyoid bone as are the hyoglossus and geniohyoid (Fig. 3). Beneath the hyoglossus muscle the lingual artery may be clamped, cut, and tied (Fig. 4). Attention is then given to the infrahyoid region, where the sternohyoid, omohyoid and thyrohyoid muscles are severed near their attachments to the bone. The superior laryngeal

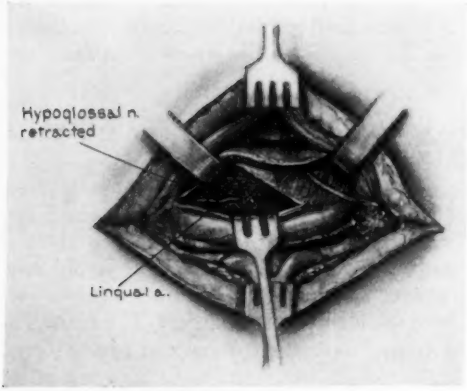

Fig. 4.—Ligation of the lingual artery beneath the hyoglossus muscle may be done more posteriorly than is shown.

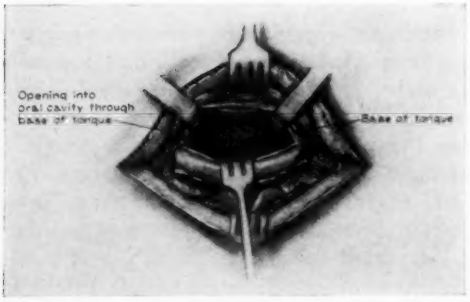

Fig. 5.—Entry to the pharynx is through the base of the tongue on side opposite lesion.

nerve is then carefully picked up and retracted backward permitting dissection of the greater cornu of the hyoid bone (Fig. 2).

One can now enter the pharynx easily through the base of the tongue, on either side well away from the vallecula and transect the base so that the corpus of the tongue recedes anteriorly into the mouth (Fig. 5). With the nerves isolated it is surprising how much of the base of the tongue can be resected without compromising the mobility of that organ. The epiglottis and the valleculae are then easily seen and the rest of the procedure can be carried out under direct vision, with section of the epiglottis at its base and of the pharyngo-epiglottic ligaments laterally to meet the tongue incision and permit removal of the specimen *en bloc* including the hyoid bone (Fig. 6).

Sutures uniting the cut edge of mucous membrane of the tongue with the cut edge of mucous membrane in the larynx effect a complete transverse closure (Fig. 7). A reinforcing layer of Lembert sutures is placed over the line of mucous membrane sutures. Flexion of the head permits union of the infrahyoid muscles with the mylohyoid and the hyglossus. The platysma and skin are closed separately in the usual manner.

Rethi[12] has expressed concern over sagging of the larynx if the suture lines give way, and has devised a method of sectioning the hyoid bone with certain of the muscles attached. He reunites the severed ends of the hyoid at the close of the procedure using drill holes in the cut ends for this purpose. This complication, i.e. separation of larynx from tongue, has not occurred in my cases. With proper approximation and reinforcement of the mucous membrane sutures plus adequate chemotherapy one need not expect a breakdown of suture lines. Rethi, dealing with lingual thyroid in both cases, expresses more concern over the integrity of the hyoid bone than he would have in the presence of malignant disease.

One may argue that this surgical method of handling carcinoma arising in the valleculae can apply only to very small lesions. Using these two cases as the basis for such a thesis, I would have to agree. Yet the postoperative resilience of these patients has been so impressive that I think the method deserves further exploration. We may well find that extirpation of a considerable portion of the base of the tongue is not incompatible with normal speech, respiration and deglutition provided the essential nerves remain intact.

SUMMARY

Early epidermoid carcinoma arising in the valleculae may be difficult to discern if the curved tip of the epiglottis refuses to leave

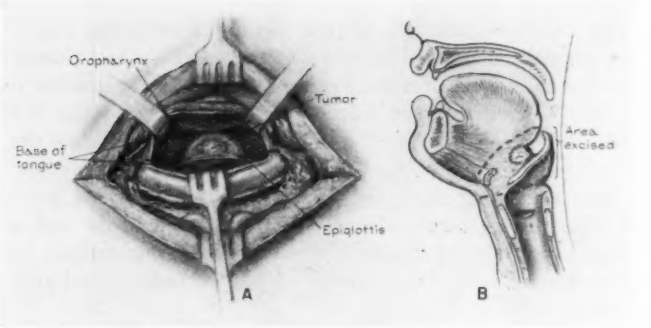

Fig. 6.—The tongue recedes upward and forward, permitting direct inspection of the tumor and the structures to be excised. The infrahyoid incision is shown by the dotted line in A. B demonstrates the volume of tissue that is resected.

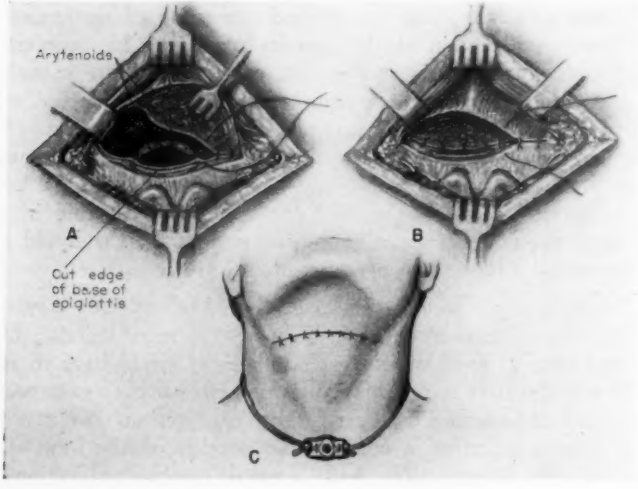

Fig. 7.—Closure of the pharynx is accomplished in layers. The base of the tongue is sutured into the larynx and this suture line re-inforced as shown in B.

the base of the tongue during the mirror examination. In the presence of hypopharyngeal discomfort, especially burning on ingestion of tart fluids, thorough inspection of the valleculae becomes essential and may require displacement of the tip of the epiglottis away from the tongue with a hook, under local anesthesia.

On routine laryngeal examination of office patients, occult valleculae occurred in 18.7%, or nearly one-fifth of those permitting examination.

The histories of two cases of epidermoid carcinoma of the vallecula illustrate the delay which may ensue if this hidden valley is not inspected.

Anterior transverse pharyngotomy proved an effective way of handling these cases. This experience, in the author's opinion, warrents further trial of this approach and technique until its limitations become apparent.

15 WASHINGTON ST.

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LXXII

SURGERY IN CARCINOMA OF THE HYPOPHARYNX

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Prior to the past decade surgery was seldom attempted for carcinoma of the hypopharynx because few patients could be prepared physiologically to withstand the extensive surgery required. Furthermore, the morbidity and mortality were so high that few surgeons cared to take the risks. Thus, almost since the advent of irradiation therapy, this modality has been the chief form of treatment for carcinoma of the hypopharynx. The results as measured in five-year survivals have been uniformly poor. The best results claimed have been 13 per cent. Martin, with the largest series reported, had only an 11 per cent cure rate. Del Regato, though an avid proponent of irradiation therapy, stated "the accomplishments of x-ray are rather poor; any effort (surgical) to improve these results is welcomed."

A frequently underemphasized but significant factor is the severe reaction experienced by the patient undergoing adequate irradiation therapy. Dryness of the mucous membranes, local pain, painful and difficult swallowing, pooling of secretions with spill-over into the larynx, probable tracheotomy, and possible gastrostomy are severe debilitating factors in an already weakened patient. Some radiologists have coined the term "radical x-ray therapy" for cancerocidal dosages. Not that adequate therapy is radical, but by so designating it, the connotation of possible harm, even death, is called to the physician's attention.

The great advances within the past decade in the adjuvants of surgery, anesthesia, parenteral medications, fluids, and foods, the ease

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Presented before the meeting of the Chicago Laryngological and Otological Society, February 1, 1954.

of transfusions, and the various antibiotics, together with the uniformly poor results of irradiation therapy, have encouraged a return to the surgical management of carcinoma of the hypopharynx.

Surgery of carcinoma of the hypopharynx follows the same principles as surgery for neoplastic tumors elsewhere in the body: wide-field excision of the primary tumor-bearing area along with the regional lymph-bearing area. Many names have been ascribed to this principle of procedure: mono-bloc, en bloc, commando. The surgery consists of unilateral or bilateral radical neck dissection in continuity with a laryngo-pharyngectomy, planned on individual needs to also include as much of any of the midline viscera of the neck, tongue, thyroid gland, trachea, and esophagus as may be required.

Experience has shown that surgery is possible in nearly all cases. The secondary extension of the tumor is a more important determining factor than is the extent of the primary lesion. That is, the size of the primary, its oral or glossal spread, or the lateral extent of the primary are of less importance in determining the feasibility of surgical excision than is the presence of metastases. If metastases are present, the operability of these metastatic areas is the deciding factor in the final choice of the method of treatment. There are cases of carcinoma of the hypopharynx which cannot be saved, being beyond all possibility of salvage when they present themselves for care, no matter what method or methods are used. Some of the contra indications to surgical intervention are:

1. Metastatic spread to vital structures.

2. Direct extension of the tumor into the posterior musculature of the neck.

3. Pulmonary or other distant metastases.

One of the difficulties in the evaluation of any method of therapy is the confusion rampant in the naming of anatomical sites and extent of the lesions described in the literature. The pharynx is an area and not an isolated structure. Our chief concern is with the hypopharynx, which is that portion extending from the esophagus up to the level of the hyoid bone. Because of the abundance of the lymphatic structures, lymph node metatases occur early and can be anticipated in nearly all cases. On this fact is predicated two basic principles of the surgical procedure: 1) The epiglottis, the pre-epiglottic tissue, and the hyoid bone are always removed, and 2) radical neck dissection is done on the side of the lesion whether nodes are palpated or not.

The only exception to the latter premise is in the event of a midline lesion with no palpable nodes. In midline lesions metastatic adenopathy may appear on either or both sides and therefore the radical neck dissection is deferred until adenopathy appears.

Before discussing specific types of cases, the final and very important phase of tumor work of this area warrants discussion: that is the rehabilitation phase. Elimination of the tumor leaves the patient voiceless, with limitation of shoulder motion due to partial denervation of the trapezius, with a tracheostomy, and often with a pharyngostomy and esophagostomy. The tracheostomy rarely poses a problem and few patients have to wear a tube beyond the immediate postoperative period. Bucco-esophageal speech is learned by most patients. Some have their problems with this chiefly those persons of advanced age who have difficulty in forming new patterns of speech or those in whom extensive tissue removal leaves an insufficient "vibrator."

The types of cases can conveniently be placed into three general groups based upon the surgical requirements for removal of the primary with an adequate margin of safety:

Group I. Those in which enough of the pharyngeal mucosa can be safely salvaged to enable primary closure and restoration of continuity of the food passage.

Group II. Those in which the extent of the lesion requires removal of all but a small posterior strip of pharyngeal mucosa. This strip is incorporated in the skin flaps in such a manner as to form a trough connecting the stomata of the pharynx and the esophagus and does aid the ultimate closure.

Group III. Those in which the entire hypopharynx must be extirpated. The skin flaps form the new vertebral covering.

In both Group II and III cases secondary closure with the formation of a new food passage must be carried out. We have been fortunate in having the guidance and suggestions of Dr. G. Kenneth Lewis of our Department in the reconstruction phases for many of these patients. Prior to this closure feeding is carried out by means of the esophagostomy, and it may be emphasized that the care of an esophagostomy is easier than that of a gastrostomy. Feeding is simpler, and the care of the surrounding skin presents no special problems.

Details of the surgical procedure are described elsewhere and here we will only stress certain aspects. Wide exposure of the field cannot be over-emphasized for adequate surgery. The neck disection is done

before the laryngo-pharyngectomy phase of the operation to create the mono-bloc. The thyroid lobe on the affected side is included in the resection. The strap muscles are severed near their distal insertions, there being no advantage in sparing the muscle mass. The trachea is always transected at or below the level of the first cartilaginous ring, and even in restricted lesions no attempt is made to salvage the mucosa of the post-cricoid area; it is resected without interrupting its attachment to the cricoid cartilage. Because the entire extent of a tumor is not always appreciated under indirect or endoscopic visualization, direct inspection is made through an incision in the unaffected lateral pharyngeal wall just prior to excision so that an adequate margin of safety is maintained under direct vision as the laryngo-pharyngectomy is completed.

SUMMARY AND CONCLUSIONS

Irradiation therapy of carcinoma of the hypopharynx has not proved successful. Numerous large series of patients treated in this way and reported over a long period of time have shown uniformly disappointing results. Recent advances in medicine have permitted a return to surgical management of these tumors with the promise of a much higher percentage of five year cures. Therefore, we feel that surgical management should be given a much more extensive trial by the men in our specialty to provide the patient with the greatest chance for survival. But survival alone is not our only objective. We feel that the surgical management will also provide the best opportunity for early rehabilitation of a useful person. We cannot cite five year survival figures in our own series but can only say that we are encouraged by the early postoperative results and anticipate a significant improvement in the ultimate outcome.

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LXXIII

JUVENILE NASOPHARYNGEAL FIBROMA IN STATE OF REGRESSION

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Juvenile nasopharyngeal fibroma in state of regression was observed by several clinicians: Shaheen,[1] Martin,[2] Figi,[3-4] New[5] and others, but its clinical and pathological features, different in many respects from those of the same tumor in state of active growth, were not sufficiently stressed by any of them. The case described in this paper was admitted to Veterans Administration Hospital on December 16, 1953. After presenting my paper at the Tumor Board of the Veterans Administration and at the Eye, Ear, Nose and Throat Staff Meeting at the Presbyterian Hospital in Albuquerque I came across Dr. Stephen S. Sternberg's[6] excellent presentation of Martin's cases of juvenile nasopharyngeal fibroma from a pathological standpoint published in the January 1954 issue of Cancer. I will return to this paper later in my discussion.

Juvenile Nasopharyngeal Fibroma, or Angiofibroma, does not belong to common tumors and presents numerous clinical and scientific problems. After many decades of research and trial of various therapeutic methods in this country and abroad, we still encounter highly diversified views about its etiology and treatment among outstanding scientists and clinicians.

The term "Juvenile Nasopharyngeal Fibroma" was first suggested by Chaveau[7] in France in 1906. Before this time, since the days of Hippocates[8] all pending tumors of the nose and the nasopharynx were described as polyps. As early as 1847, Chelius[9] in England stated that fibrous nasal polyps commonly occurred in persons about the time of puberty. Legouest[10] in 1865 called attention to the selectivity of this tumor for males, and Gosselin,[11] in 1876 noted its tendency toward spontaneous regression after sexual maturity.

INCIDENCE

Figi[3, 4] of the Mayo Clinic, who has probably seen and treated more cases of juvenile fibroma than anybody else, reported 114 cases in 40 years' experience. Another large series of cases (58) were re-

ported by Shaheen[1] in 1930, but Figi and Martin and their associates are very skeptical about the correctness of Shaheen's diagnosis since he offered no histologic or other corroborating data and the age extremes of two years to seventy years, definitely exclude juvenile nasopharyngeal fibroma in some of them. Härkönen and Malmio[12] reported 31 cases. Martin[2] and his associates observed 29 cases in a period of twenty years and reported them in 1948. Numerous isolated reports describing from one to seven cases have been found in the medical literature. I have diagnosed and taken care of six cases during 32 years of my practice.

ETIOLOGY

The etiology of this neoplasm is still an unsolved problem. At least three theories are advanced. The theory first promoted by Verneuil[13] in 1878 and later supported by such outstanding pathologists as Bensch,[13] Ewing,[14] and others, states that the tumor develops from the perichondrium of the embryonic occipital plate which forms the basal portion of the occipital bone, body of the sphenoid, medial plate of the pterygoid process, and bone around the foramen lacerum. Till 25 years of age, the basilar portion of occipital bone is connected with the body of sphenoid by fibrous cartilage, which after this age is completely ossified. Several objections were raised lately against this theory; it does not explain the sexual selectivity, does not consider the angiomatous element of the neoplasm, and this tumor does not contain cartilage or bone as its integral parts which should be expected as a product of perichondrium.

The second theory is advocated by Brunner[15] and supported by Francis Lederer[16] in the newest edition of his textbook. According to this theory, juvenile fibroma originates from basilar fascia or pharyngeal aponeurosis of the superior constrictor muscle which covers the posterior wall and roof of the nasopharynx. Some of the objections like sexual selectivity, for instance, raised against Verneuil's theory are applicable also to this theory.

The third theory forwarded by Martin[2] stresses three essential facts for histogenesis of this tumor: 1) Angiomatous state of the initial stage of the neoplasm; 2) sex relationship, and 3) age incidence. Juvenile fibroma starts developing from a very rich vascular meshwork of this area at the age of sudden increase of androgenic stimulation. At this initial stage it displays histologically a picture of an angioma occasionally cavernous in type with a very scant fibromatous element. Martin is the first who states that this tumor is limited to males and all his patients are males. Although the second series of Figi[4] consists entirely of males, his first series contains five

females, and he is still of the opinion that this tumor can occur occasionally in females. St. Clair Thomson and Negus[17] also state that this neoplasm occurs rarely in females, and display in the last edition of their textbook a picture of a negress with characteristic "frog-face" appearance and tumor protruding from both her nostrils. Finerman[18] of Los Angeles described a case of juvenile fibroma in a 13 year old girl, confirmed by histological examination by three reliable pathologists. Diel[19] reported a case in a woman and there were several more cases described and confirmed by histo-pathological examination. Martin, in my opinion, is only partly right. I would like to suggest a little correction of his theory: the tumor is not sex-bound, but androgen-bound. This correction would fully explain why the tumor occurs predominantly in males and why it can occasionally occur in females. It is a well known fact that some small amount of opposite sex hormone is produced by individuals of both sexes. In 1940 Beall[20] isolated estrogenic hormones from the testes of the stallion, in 1943 Ruzicka and Prelog[21]—from the testes of the hog, the same was repeated by Haines and associates[22] in 1948. In 1952 Goldzieher and Roberts[23] isolated estrogens from human testes and Schaffenburg and McCullagh[24] isolated them from human sperm. The same pertains to the female: In 1940 Callow and Callow[25] isolated androsterone 1, 3 mgrs per liter, etiocholanol-3 alpha-one-17 1, 3 mgrs per liter and dehydroepiandrosterone 0.2 mgrs per liter from the urine of normal women. In adrenal cancer the concentration of dehydroepiandrosterone is enormously increased.[26] The level of androgens in females during puberty is not constant but fluctuates to a great extent. Until normally balanced interplay between the ovarian hormones on one side and the pituitary gonadotropins and corticoadrenal hormones on the other side is established the formation of androgenic hormones may for some time well exceed the upper limit of normality. This unbalance between the production of androgenic and estrogenic hormones in the female during puberty, although present for a short period, may cause initiation of juvenile fibroma in females. I think this supposition is worth consideration and further studies. When fluctuations of sexual development have ended and full maturity is reached, approximately at the age between 20 and 25, the spontaneous regression of the tumor starts. This fact definitely proves the relationship between this growth and sexual development.

INCIDENCE AND ANATOMY

The onset of symptoms, according to Martin,[2] occurs between the ages of 7 and 19, average at 14, and according to Figi,[4] between 9 and 17, average 13½ years.

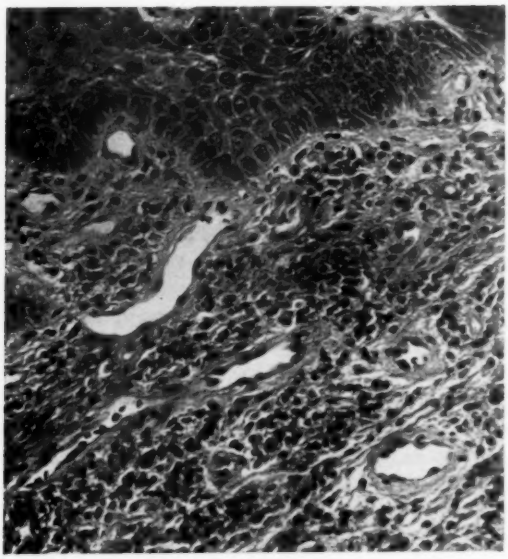

Fig. 1.—Photomicrograph, juvenile fibroma. The surface of the tumor is covered by a hyperplastic stratified squamous cell epithelium (metaplasia) exhibiting areas of dyskeratosis in the basal layers. The underlying stroma consists of two characteristic elements: fibrous and vascular. The fibrous stroma near the surface demonstrates young fibroblastic proliferation, the cells and their nuclei are of different size and shape. The thin-walled vessels of various caliber are round or elongated; their walls consist of a single flat endothelial layer only; a little deeper (at the right side of the picture (the lumen of the vessels is narrowed probably due to stromal compression.

Nasopharyngeal fibroma is a tumor of different sizes and varied shapes—ovoid, club-shaped, smooth or lobulated and often with extensions into the nasal cavities. It is usually sessile with a broad base but occasionally pedunculated attached to the vault of the nasopharynx, its roof, and the upper part of the posterior wall. The surface, if not traumatized, is covered by mucous membrane highly injected, deep red in color in younger subjects and pale pink in older. The consistency varies, dependent on the stage of development. In the early stage, the tumor is soft, later rubbery, then, when the fibrous element predominates, cartilaginoid; in the stage of regression, the consistency varies in different parts of the same tumor, may be, in some places soft, edematous, even friable. On section, the color of the

neoplasm ranges from pinkish white to greyish yellow, to reddish brown, in places it shows translucent or homogenous tissue. Vascularity is dependent upon the age of the patient.

MICROSCOPIC PATHOLOGY

The usual histologic pattern consists of connective tissue, stroma and vessels whose thin walls are devoid of contractile elastic tissue and muscles. The younger the tumor the greater the number of vessels and cavernous lakes, the scarcer the stroma and the more immature the connective tissue cells forming the stroma; they are spindle and star-shaped fibroblasts, sometimes so numerous as to suggest angiosarcoma. The older the tumor, the smaller is the number of vessels, the more predominant is the fibrous stroma, the more mature are the fibroblasts forming it, especially in the central areas of the tumor. Many vessels in the central areas are obliterated either by thrombi which very often undergo hyalin degeneration, or by gradual narrowing of their lumina due to "stromal compression" (Sternberg); in places the lumen completely disappears, only a double row of endothelial cells opposing one another are found. In the state of regression myxomatous changes in varying proportion and sclerosed areas of compact acellular fibrous tissue neighbor with areas of necrosis and hyalinized stroma. Foreign body giant cells are often seen near the surface. At the periphery the tumor never loses its fundamental histological pattern: the rich vascular network and young connective tissue stroma. The tumor does not possess a true capsule and is covered by pharyngeal mucosa; in places the mucosa is ulcerated or shows squamous cell metaplasia. The tumor does not undergo malignant changes; none occurred in Martin's series, and among 114 of Figi's patients one was suspected of malignant changes—a man 20 years of age from whose nasopharynx, nasal fossa, and sphenoid sinus, a large recurrent vascular fibroma was removed. In a period of three and one-half years after the operation, he experienced right frontal and parietal headaches with gradual development of right VIth nerve paralysis and complete bilateral blindness. The author himself did not exclude an inflammatory process as the sole cause of the symptoms in this case. Single cases reported by Shaheen,[1] Jackson[27, 28] and Dabney[29] do not present adequate and convincing evidence in the opinion of Martin, Figi and their associates.

SYMPTOMS AND CLINICAL COURSE

The symptoms depend upon the size of the tumor, the direction of its expansion, the state of its surface, i.e., whether it is intact or traumatized and inflamed, and upon the stage of its development. Among the early symptoms of juvenile fibroma is nasal obstruction.

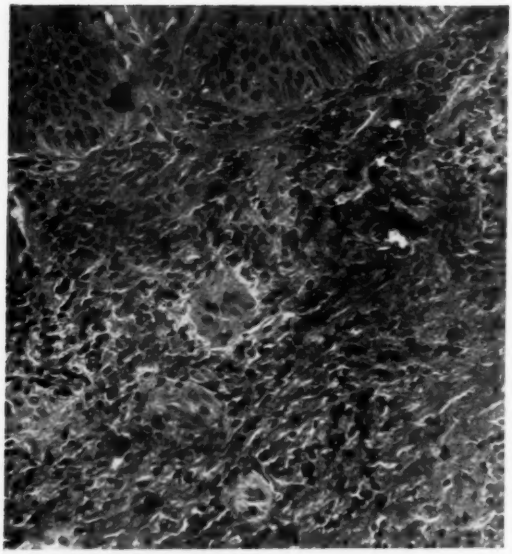

Fig. 2.—Several multinucleated giant cells are seen not far from the periphery of the tumor, numerous mononuclear cells, as well as plasma cells, are scattered throughout the surrounding region.

When the tumor completely obliterates both choanae, the patient becomes a mouth-breather, loses his sense of smell, his taste is also affected, his voice changes, speech becomes muffled, indistinct-rhinolalia clausa develops, and his face assumes the typical expression. The growth expanding downward and laterally, may be seen protruding below the soft palate margin on examination of the pharynx, may obstruct tubal ostia and produce, consequently, catarrh of the middle ears with accompanying symptoms of deafness, tinnitus, and occasional vertigo.

When the tumor expands straight forward through the nasal cavities, an edematous, sometimes partly necrotized mass, may be seen protruding from one or both nares. Expansion of the tumor upward may cause elevation of the orbits with consequent proptosis of the eyeballs, and typical frog-face deformity is produced. In some cases the bulky mass of the tumor by pressure necrosis either gradually pushes the thinned medial wall of the antrum lateralward or invades the sinual cavities, and by further expansion forward,

causes facial deformity, usually unilateral. Goldsmith[30] described a case of juvenile fibroma which eroded the base of the skull.

Among early symptoms is nosebleed from the nasopharynx. Epistaxis may be infrequent and moderate, but very often nosebleeds occur at short intervals, are profuse and necessitate nasal packing. Nasal packing causes trauma and necrosis, followed by more profuse bleeding. In this way a vicious circle is established. In consequence, the patient may become extremely anemic and require blood transfusion. Local necrosis of the tumor results usually in sepsis which spreads into nasal sinuses and middle ears.

Besides all the described symptoms, the patient suffers mentally, being socially degraded and economically handicapped.

Diagnosis is made on the basis of history, appearance of the tumor, its location, the age and sex of the patient, by differential exclusion of other entities, by radiographic studies and finally by biopsy. The examination is performed with the nasopharyngeal mirror. Sometimes the tumor may be seen, on examination of the pharynx, protruding below the margin of the soft palate, by anterior rhinoscopy, by nasopharyngoscopy, and by cautious manual palpation.

Malignant tumors of the nasopharynx are uncommon in children and adolescents and can be easily excluded because of a different appearance on examination and x-ray films, more rapid course, and early metastasis in lymph nodes. If possible, biopsy should be performed. Choanal polyps are usually multiple, pedunculated, pale, edematous and occur mostly in individuals suffering from allergy. Adenoids do not present any diagnostic difficulty to a laryngologist. Martin mentioned three rare conditions which he came across in his practice and which clinically resemble juvenile nasopharyngeal fibroma in every way. They were: chondroma, tuberculosis of the retropharyngeal lymph node and olfactory aestesioneuroblastoma in the postnasal space. Cellular fibroma of the nasopharynx resembles clinically juvenile angiofibroma. Although very desirable, all writers on juvenile fibroma have some objection to biopsy. They advise extreme caution and point out the limitations. The threat of profuse, hardly manageable hemorrhage is so great that many clinicians abstain from biopsy in most of their cases. Figi avoids it in clear cut cases; only a little more than one-half of his cases were biopsied. Martin displays the same attitude. Biopsy has been performed always in hospitals, with satisfactory anesthesia and with all facilities ready for the control of profuse hemorrhage, and for blood transfusions. Munson[31] stresses the fact observed by him and others that the ligation of

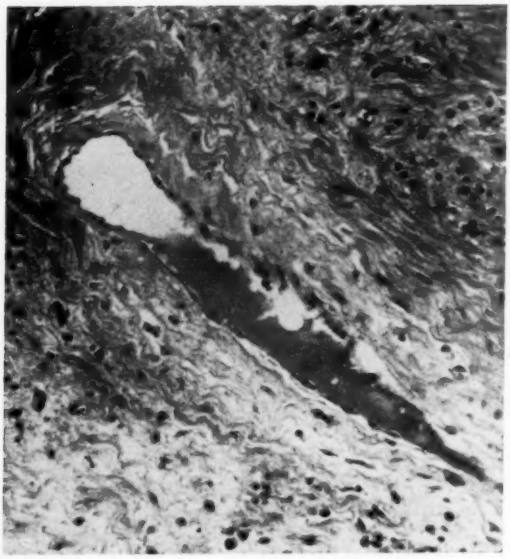

Fig. 3.—A large narrow vessel obliterated by a hyalinized thrombus is surrounded by loose myxomatous stroma.

both external carotids does not seem to lessen the tendency of the tumor to bleed during manipulation. Brunner's[15] studies in 1942 proved that the angiomatous vessels of the juvenile fibroma communicate only with veins of the normal surrounding tissues but not with the arteries. Taking into consideration the rich venous supply of this region—like the pterygoid plexus which communities not only with the anterior facial veins but also with the veins of the cranial cavity by the way of the superior ophthalmic veins and with the cavernous sinus by a network of emissary veins running through foramina ovale, lacerum, and Vesalii—the tendency to bleeding and difficulty of controlling it are fully understandable.

TREATMENT

Although treatment of juvenile nasopharyngeal fibroma, due to better understanding of the character of the tumor, has undergone considerable change during the last few decades and generally has shifted to a more conservative approach, nevertheless, the methods used by clinicians, both surgical and conservative, still differ con-

siderably. Unanimous opinion now prevails that treatment is applied only in those cases with definite symptoms. There is no necessity of any treatment in cases of advanced regression of the tumor even when less distressing symptoms are still present. The conservative treatment consists of androgenic hormones, anti-anemic measures, vitamins, radiation therapy by x-ray and interstial radiation. Different surgical methods by various approaches and electro-surgery are in use. Both conservative treatment and surgery are applied separately in some cases, but mostly they are used in combination. The encouraging fibromatogenic and antifibromatogenic experiments with steroid hormones by A. Lipschutz and his co-workers[32-35] in Chile had stimulated many clinicians to exploit the new agents in treatment of fibromas. Martin hoped for a time to be able to control entirely nasopharyngeal fibro-angioma by endocrine therapy alone. This has not proved possible and only in one case of an 18 year old male (who was treated previously with limited benefit by radiation, intramuscular application of 25 mgrs of testosterone proprionate every second day during one month) showed a marked change of his secondary sex characteristics and rapid disappearance of the tumor. The use of hormonal therapy may be justified only in cases with definitely established retardation in sexual development. Martin, however, as seen from Sternberg's report, used androgenic treatment injudiciously in many cases. He even applied this kind of treatment to a 36 year old patient and certainly, as should be expected, the therapy was followed by tumor increase; he experienced the same result in another case. An abnormally high as well as low level of androgenic hormones disturbing the harmonious action of other hormones participating proportionally in the interplay may be one of the predisposing factors of the tumor's histogenesis. The work of E. A. Edwards,[36] S. R. M. Reynolds,[37, 38] E. J. McGrath,[39] and their associates on the use of androgens in hypogonadal males has revealed an increase in the cutaneous blood flow. If a similar situation exists in the nasopharyngeal mucosa of the sexually retarded patients suffering from juvenile angiofibroma the rationale of testosterone therapy may be highly questionable. Figi does not mention in his reports hormonal therapy at all. Penha[40] reported a dramatic recovery from a profusely bleeding juvenile angiofibroma in a 12 year old boy by application of an infusion and aqueous extract of *urtica membranacea* by mouth together with daily blood transfusion. Penha used with success injections into the tumor concomitant with intramuscular and intravenous injections of purified juice of the same plant in another less severe case of nasopharyngeal fibroma. He did not, however, present any microscopic evidence supporting his diagnosis.

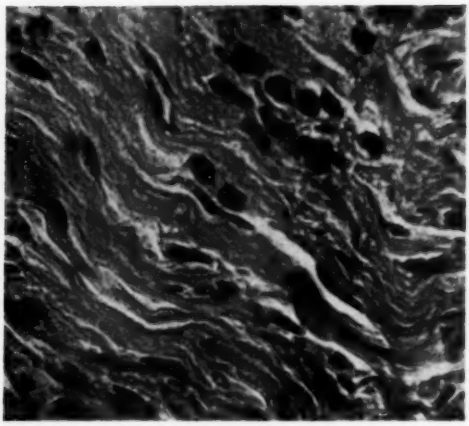

Fig. 4.—Detail showing the young fibroblasts assuming a more uniformly elongated shape in somewhat deeper areas and still farther (upper left) changing into acellular collagenous coarse fibrous tissue.

Radiation therapy either by roentgen rays or by interstitial radium application in the form of gold radon implants, seeds, or needles in well established dosages are the most useful methods. The proper dosage in these cases is of great importance in view of the proximity of the germinal centers of growing bones; overdosage may cause permanent deformity. Martin suggests x-ray therapy not to exceed 1500 r. in divided doses through circular portals 5 cm in diameter over each maxilla. The dose through the palate may reach 2500 r. through portals 3-4 cm in diameter. Samuel Henley[41] and associates reported three cases treated successfully with roentgen rays; only 1100 r. were applied in each case. It is understood that only the angiomatous element of juvenile fibroma is radio-sensitive, not its fibrous stroma. Radium therapy, by means of implants may be applied to less vascular and bulky tumors, not exceeding five cm in diameter. This treatment can be combined with roentgen rays, and hormonal therapy. Seeds in doses of 5 to 6 mcs can be inserted through the nasal cavity, transorally through the nasopharynx or through the soft palate. Twenty years ago when the character of this tumor was not fully understood and its complete eradication was thought necessary, Martin experienced several serious complications due to overdosage of interstitial radiation in three cases; in one—necrosis of the hard palate, in two extensive osteonecrosis extending

into the sphenoid bone; both of the latter patients succumbed to brain abscess.

SURGICAL METHODS

The indication for surgery in cases of juvenile fibroma has narrowed for the last two decades only to the large tumors with marked facial deformity, progressive destruction of the maxilla or other adjacent structures by pressure necrosis, and to those tumors which bleed frequently and profusely or show local sepsis giving rise to sinusitis or otitis media. In all these cases preliminary irradiation and hormonal treatment, if possible, is applied to lessen the hazard of hemorrhage during surgery. The disfiguring hazardous avenues of approach like Moure's lateral rhinotomy, Ollier's total rhinotomy and Langenbeck's and Denker's operations have long been discarded. Transpalatine approach, although disapproved by most of the surgeons, still is used by some. Brown[42] reported six cases of juvenile fibroma successfully removed through a palatine approach with intratracheal anesthesia. Preliminary ligation of one or both external carotid arteries was necessary in most of his cases. Alfredo Alcaino[43] described his own technique for nasopharyngeal tumors and presented six patients successfully operated upon by his method. It consists of bilateral subperichondrial and subperiosteal elevation of the entire septal mucosa down to the middle of the nasal floor; the whole septum from the anterior edge of quadrangular cartilage which is divided from the upper lateral cartilages through its bony part to the posterior margin of the vomer, is fractured and moved to the side where the nasopharyngeal tumor is less adjacent to the lateral wall. He also infracts on both sides the medial and inferior turbinates and presses them toward the lateral wall of the nasal cavities. This procedure increases the operating space considerably. After removal of the tumor from the nasopharynx the turbinates and septum are retracted to the normal position. At this time, deviation of the septum may be corrected. It is impossible to obtain wide surgical exposure of the field, since the area itself has many natural furrows, crevices with adjacent cavities and sinuses usually invaded by the tumor, and the operation is very often accompanied by profuse bleeding. Therefore, complete eradication of the growth by surgery in most of the cases cannot be accomplished. All these facts are responsible for frequent recurrences of tumors operated upon. Nevertheless, where strong indications arise, surgery is performed in a hospital, combined with all precautionary measures such as preliminary therapy, ligation of the external carotid arteries, and blood at hand for transfusion. Among the most generally used methods is wire snaring through the anterior nares or simple evulsion by digital manipulation through

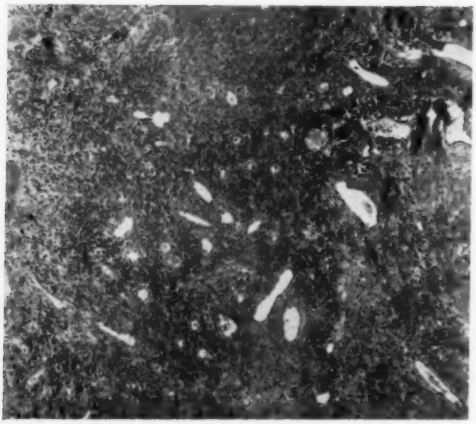

Fig. 5.—Numerous thin-walled vascular channels of different caliber and shape are scattered in fibrous stroma showing various degrees of degeneration: myxomatous, hyalin, sclerotic, with a rich invasion of chronic inflammatory cells.

the mouth in cases of smaller tumors. In large tumors Martin[2] uses an approach through the alveolar process anteriorly just to one side of the midline. He extracts 4 or 5 teeth if they are present, removes the alveolar process above the extracted teeth, exposing widely the antrum and nasal cavity on this side, then he removes the entire antronasal wall. This gives him a fairly wide exposure of the nasopharynx with adequate accessibility to the ethmoid and sphenoid sinuses. He assures us that the operation is followed by surprisingly little disability when a dental prosthesis completely covers the defect. The remnant of the tumor should be treated postoperatively by radiation. Figi[4] states that the most satisfactory treatment for nasopharyngeal fibroma is a combination of electrocoagulation and interstitial radiation. These measures may be used through the nares, transorally, through the nasopharynx, and through a transantral approach. In this approach he is less radical than Martin; he leaves the alveolar process alone, performs a Caldwell-Luc operation removing most of the anterior wall and part of the naso-antral wall.

Let me state Martin's conclusion drawn from a thorough study of his cases. "It is plain that many of the most serious complications are brought on primarily by aggressive and ill-conceived therapeutic measures, such as poorly planned attempts of surgical removal with-

out preliminary arterial ligation and inadequate exposure of the nasopharynx, and secondarily, by unduly vigorous endeavors to control further growth by radiation therapy. In such cases the patient would have been much better off without treatment whatever." I agree fully with this statement.

The case, which I am going to present, belongs to those fortunate ones who despite having developed a large tumor and having suffered very much from it for many years, has not been examined by any specialist before me. He was seen from time to time by a general practitioner who had not diagnosed the condition and had prescribed some nose drops. This situation lasted until the tumor had started spontaneous regression and thus the patient's condition started improving. Who knows what would happen to this man (afflicted also with rheumatic heart disease, rheumatic valvulitis, aortic insufficiency, and left ventricular enlargement) if he were treated and operated on by an energetic aggressive specialist at the summit of the tumor's development? In the opinion of the Medical Staff of this hospital, the patient's heart condition requires constant vigilant care, and any infection or operative procedure, even a minor one, can aggravate an old valvular lesion.

REPORT OF A CASE

A 31-year old white male was completely unaware of the presence of a tumor in his nasopharynx until it was found by the ward physician in our hospital on routine general examination on December 20, 1953.

The patient stated that from the age of 12 he had suffered from almost constant pressing headache around the orbits; the pain was sometimes so severe that he felt as if his eyeballs were coming out from the orbits. The headaches were worse on the right side. Until approximately the age of 16 he did not notice any other symptoms. In 1937 he moved from the State of Washington to New Mexico. His headache lessened considerably but he noticed that his voice became indistinct so that people often could not understand him. At the same time he noticed impairment in hearing, especially on the right side. The last two symptoms had gradually increased until the age of 20 when the condition became stationary. At that time, in 1944 and 1945, patient's hearing was very bad and he could not breathe completely through the nose; both sides were obstructed continuously. From time to time he had suffered from moderate epistaxis and more profuse bleeding from the nasopharynx. The breathing through the nose and the improvement in hearing started about the end of the last year, and not only the patient but also his

wife and people around him had noticed great improvement in his hearing. He had not been losing weight.

From the time of the patient's admission on December 16, 1953, until May 7, 1954, he was confined to bed, put on cardiac regime and treated, among other medications, with quinidine because of premature ventricular contraction. On May 7, the streptolysis titre was 250 units and the sedimentation rate 17 mm (22 mm on admission) which gradually went down and on the 12th day reached 11 mm with a normal white cell count. The patient's general condition improved greatly. The etiology of aortic insufficiency with valvulitis and left ventricular enlargement in this case was rheumatic in origin.

Some dryness and small thin crusts were seen in the anterior aspect of both nasal cavities. Anteriorly the septum was moderately deviated to the left. Before and after shrinkage of the nasal mucosa, adequate breathing space was present on both sides. In the posterior third of the right middle meatus a round pink greyish mass of smooth surface was seen. A probe moved around it easily toward the nasopharynx.

Transillumination of the nasal sinuses was negative. X-ray films revealed the contour of the tumor in the nasopharynx and also a slight haziness of the maxillary sinuses, more pronounced on the right side.

The mouth showed no important changes. The tonsils had been removed at the age of 6. Behind the right posterior pillar a roundish smooth dark red mass protruded for about .5 cm.

Posterior rhinoscopy revealed two round smooth surfaced masses protruding, the larger of the two, about the size of a plum on the right side, the other higher, attached to the first, about the size of a small cherry on the left side.

Palpation revealed a lobulated tumor of various consistency, from very soft to firm cartilaginoid, attached on a broad basis to the vault of nasopharynx. After palpation there was a very slight admixture of blood with the mucous.

Both external auditory canals were normal. Both eardrums were slightly retracted, the right more than the left. The light reflex on the right side was broadened and shortened, and the whole eardrum was slightly dull.

Audiometry revealed normal hearing in both ears to and below 2048 dv. At 4096 dv there was a dip of 20 db on the left and 35 db on the right. On this side the tumor had covered the entrance

of the eustachian tube for a considerably longer period than on the left side.

Cervical lymph nodes were not palpable.

DIAGNOSIS

The diagnosis was adolescent hemangiofibroma in the state of regression. An exploratory puncture of the tumor with a 19 gauge needle 3 mm deep was performed and bleeding was watched. The bleeding was minimal and lasted for about 11 minutes. I made the exploration having in mind a future attempt at biopsy with all precautionary measures against hemorrhage.

Further observation, without any treatment of the tumor, was suggested by me.

On May 27, 1954, under local anesthesia with 2 per cent pontocaine and submucosal injection of the tumor with 2 per cent procaine about ¾ cm above its lower end, 1½ cc of 5 per cent sodium morrhuate was injected through the same needle into the substance of the tumor at the same level. About 10 minutes later the top of the tumor was biopsied with a medium-sized Hartman tonsil punch. The piece, of oval shape about 8 x 6 mm in diameter and 3 mm in thickness in its middle part was sent to the laboratory for examination. It had the consistency of cartilage. At the edges of the biopsy wound the tumor was bleeding moderately and the edges were electrocoagulated. The middle of the biopsied surface about 6 x 4 mm in diameter was smooth, dark blue in color and on pressure with a beaded probe gave the impression of a large venous sinus or cyst with a thin venous wall. The bleeding of the edges was completely controlled by electrocoagulation.

The pathological report on June 4, 1954 was as follows:

Gross: "The specimen is said to be a biopsy from a tumor in the nasopharynx and consists of a gray, firm tissue measuring 1 x 0.6 x 0.2 cm.

Microscopic: Part of one surface is covered by a broadened layer of hyperplastic epithelium exhibiting areas of dyskeratosis in the basal layers but not malignant change. The underlying stroma demonstrates varying degrees of fibrosis with early and late fibroplastic proliferation. The depths of the section also demonstrate numerous dilated small thinly lined capillary vascular channels many of which are included in the above described sclerotic process whereas others appear to be in areas of myxomatous change. Scattered throughout the regions of myxomatous change are numerous mononuclear, as

well as plasma cells. In several instances hyaline-like thrombi are present in the distended capillaries. Immediately adjacent to the keratinizing epithelium are aggregates of multinuclear giant cells surrounded by a peripheral lymphocytic infiltrate.

Diagnosis: Fibroangioma with epithelial hyperplasia and inflammatory reaction, chronic." (Wm. Hentel, M.D., Pathologist)

CONCLUSIONS

The history reveals spontaneous gradual disappearance of the most distressing symptoms after full maturity was reached. The course in the hospital with further progressive improvement without any treatment has pointed definitely to the regressive stage of the growth. The pathological picture has also shown characteristic features for this stage. Different consistency in various areas of the tumor found on palpation was due to widespread advanced degenerative processes which have taken place in the neoplasm during regression and have been followed by resorption, absorption, and shrinkage of the growth. On microscopic examination all the processes are clearly evident. The basically typical pattern of adolescent angiofibroma has been present only at the very periphery. In the deeper strata all kinds of degenerative processes of various degrees have surrounded a still intact vascular meshwork which probably, as a venous capillary system, performs the absorptive function. Still deeper the angiomatous element has been affected: the lumina have been narrowed and occluded by stromal compression, thrombi, inflammation of their walls, and the vessels have finally completely disappeared.

The patient's general health in the hospital has improved greatly. He withstood the biopsy and electrocoagulation well, had no bleeding and did not complain of any discomfort postoperatively. On May 26, 1954, he was found by the Medical Staff and the consulting physician, Dr. Albert Maisel, to be fully rehabilitated for work requiring no undue physical exertion, and the patient himself decided to take a special type of training in electronics consistent with his physical condition. He was discharged on June 3, 1954 and advised to appear periodically for a checkup.

SUMMARY

1. Juvenile nasopharyngeal fibroma, theories of its histogenesis, pathology, clinical course, and different methods of treatment are discussed.

2. Pathological and clinical features of juvenile fibroma in the state of regression are emphasized and presented in a typical cas

3. It is suggested that no therapy should be employed in cases of juvenile nasopharyngeal fibroma showing definite spontaneous regression.

VETERAN'S ADMINISTRATION HOSPITAL.

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LXXIV

ELECTRODIAGNOSTIC-THERAPEUTIC MODALITIES IN FACIAL NERVE PARALYSIS

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Conservative management of peripheral seventh cranial nerve paralysis must be based upon careful diagnosis, scientific assessment of the status of the nerve lesion, and rational therapy designed to promote the optimal return of function to the involved part.

It is the purpose of this paper to describe electrodiagnostic techniques used by the authors and to outline a rational method of conservative therapy.

Two combined technics of electrodiagnosis are considered adequate for assessing the status of the facial nerve. The Faradic-Galvanic test serves as a rough screening measure whereas electromyography is used for more precise information.

THE FARADIC-GALVANIC TEST

The faradic-galvanic test is a study of the response of the nerve and its corresponding muscles to two types of currents. This principle of electrodiagnosis was first studied by Duchenne and further developed by such men as Erb, Remak, Pfluger, DuBois, Reymond and Babinski.[1, 2] Recently more accurate tests have been described such as intensity duration tests,[3, 4] galvanic tetanus ratio, strength interval curves,[5] and strength frequency curves.[6] While admittedly these tests are more quantitative and give more information than the faradic-galvanic test, they are not adequate in themselves, are more time-consuming in their performance, and therefore do not qualify as a screening procedure when electromyographic testing is contemplated.

Detailed descriptions of the testing techniques are available in standard textbooks of physical medicine and neurology and will be

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outlined only briefly. Any generator of faradic and galvanic currents, approved by the Council of Physical Medicine and Rehabilitation of the American Medical Association may be used. A large dispersive electrode may be placed on the back, the upper arm or the hand. The testing electrode is first placed over the nerve point of the facial nerve trunk just anterior to the tragus, and the threshold value (which is the minimal intensity of current required to obtain a liminal response) is determined first for the unaffected side and then for the involved side. These values should be noted for both the faradic (induced or "tetanizing" current) and the galvanic (constant, voltaic, or "twitch" current). The upper, middle and lower branches should also be tested separately and, finally, representative muscles in each area are tested directly.

The findings in the reaction of degeneration (R.D.) are designated as: 1) No R.D.; 2) mild partial R.D.; 3) severe partial R.D.; 4) full R.D.; 5) absolute R.D. No R.D. indicates that the responses to both currents in the facial nerve and the facial muscles are present. Hypo- or hyper-excitability may be present but innervation is intact. Mild partial R.D. means there is a decrease in both faradic and galvanic response in the nerve and decrease in the response of the muscle to faradic current with usually an increased excitability of the muscle to galvanic current. Polar relations may be altered. In severe partial R.D. these changes are more pronounced. In full R.D. the nerve does not respond to either current and after an initial period of hyperexcitability (in our experience two to seven days) the muscle will not respond to faradic current at the highest intensity tolerated by the patient. The muscle responds to galvanic stimulation but with qualitative, quantitative and polar changes. Quantitatively, initially there is increased excitability, responses being obtained with as little as one-third the amount of current normally required. With the passage of time, the muscle loses its excitability and requires more than normal current. Qualitatively, the muscles respond slowly (vermicular) as compared to the brisk lightning-like normal response, and finally the polar relation may be reversed or altered. In the absolute R.D. no responses are obtained to either current in the facial nerve or the muscles.

When the test is performed at about the second or third week after the onset we have found in cases of Bell's palsy that the test has reliable prognostic significance. If the test is performed after the first month or more, it cannot be used as a reliable prognostic procedure because responses to the electrical stimuli lag behind and do not correlate with the degree of nerve regeneratoin.

A review of some routine cases seen at the Illinois Central Hospital* and Research and Educational Hospital, and examined in the Department of Physical Medicine and Rehabilitation confirmed the above statements. Table 1 shows that the cases which had the mildest electrical changes also had the quickest and most complete recovery, whereas those with profound changes had slower and less complete recovery. Case 4 was examined too late for the test to be of any significance. When there was no R.D., recovery began as early as two weeks after the onset of paralysis and was sometimes complete in five weeks. In mild partial R.D. recovery was well under way by the fifth week. In moderate partial R.D. there was approximately 90 per cent recovery in five months (Case 11) and 15 per cent recovery in the fourth week (Case 2). In full R.D. the patient may take six months to a year before recovery occurs and it is usually incomplete. Case 3 showed only 30 per cent recovery when seen 17 months after the onset. The only cases of absolute R.D. seen by us were those who had facial paralysis for over one year, usually over four years. It was assumed that complete fibrous or fatty replacement of muscle tissue had occurred in these cases. However, other changes in muscle tissue could also cause absolute R.D. These could be related to alterations in chemistry and electrophysiological characteristics.

ELECTROMYOGRAPHY

electrical properties of skeletal muscle. The techniques[8] employed in our work consist of inserting a needle electrode directly into the muscles to be studied, leading this off to a suitable amplifier, and then to an audio unit and oscilloscope.

Action potentials in the facial musculature are of shorter duration than those of the limbs or trunk.[11] Furthermore, the normal "relaxation silence" seen in other parts of the body is obtained with considerable difficulty in the face. Another peculiarity of the face is the pick up of distant activity which usually comes from the uninvolved side of the face during effort, or from adjacent muscles innervated by the normal fifth nerve. All of these characteristics confuse the inexperienced and serve to make facial electromyography truly the domain of the most experienced.

However, in skilled hands electromyography yields basic information which should give the clinician the best possible understanding of his patient. Normally, periods of electrical silence may be obtained when the patient attempts to relax or rest the face. If this

*We are indebted to Dr. E. C. Olson, Chief Surgeon of the Illinois Central Hospital, for permission to use this material.

is not obtainable, the experienced electromyographer can tell whether the action potentials seen do or do not have pathological significance. With minimal contraction effort many action potentials (normal motor units) of short duration are noted. With maximum effort a summation effect is seen and the so-called interference pattern is recorded.

If Wallerian degeneration has taken place in the facial nerve then, at rest, there are recorded the spontaneously occurring fibrillation potentials. These usually develop 14 to 18 days after the onset. Their presence indicates a poorer prognosis. With effort there will be no change in the pattern (if denervation is truly complete) except insofar as distant electrical activity from the opposite side of the face or occasionally from the masseter or temporalis may influence the pattern. Such activity has a muffled sound on the audio and the potentials are somewhat attenuated in appearance on the oscilloscope. The earliest sign of regeneration is the polyphasic nascent unit which signifies a good prognosis. This unit appears on an average of eight weeks before there is clinical evidence of recovery. Other signs of regeneration are disappearance of the fibrillation potential and the gradual appearance and increase in the number of normal motor unit potentials.

When there is physiological block without nerve degeneration electromyography reveals electrical silence during rest as well as during effort. The prognosis should be good unless the blocking mechanism becomes increasingly severe and leads to Wallerian degeneration. This condition is found when there is no electrical R.D. However, it has also been found in the presence of full R.D. and provides an example of the greater value electromyography has over the faradic-galvanic test (or other electrodiagnostic tests, for that matter).

CONSERVATIVE THERAPY OF FACIAL NERVE PARALYSIS

The therapy of idiopathic facial paralysis varies today from the do-nothing attitude of the therapeutic nihilists to the opposite extreme of performing routine surgical decompressions of the facial nerve. More recently cortisone and stellate block therapy[14] have been advocated. It should be apparent that no therapeutic measure can be evaluated without the benefit of electrodiagnostic and electromyographic testing because of the great variability of the final outcome in this disorder.[15]

Theoretically, the most logical treatment would consist of any measure or measures designed to relieve the facial nerve of the pressure effects of edema, as soon after the onset as possible. It is con-

ceivable that such treatment might minimize or arrest damage to the facial nerve and consequently result in earlier and more complete restoration of function. The greatest difficulty lies in the fact that the nerve very likely has sustained the greatest insult just prior to or simultaneous with the onset. The physician sees the patient when it may be too late to do much good with respect to relief of edema. In those cases where edema is prolonged over a long period of time decongestive measures should be beneficial, and surgical decompression would certainly be a rational measure. But this should be based upon scientific diagnosis lest many needless operations be performed.

Once paralysis of the facial muscles has set in a second objective of therapy is to prevent or retard atrophic and fibrotic degeneration of muscle fibers, and to retard the electrophysiologic and chemical changes that accompany denervation. Restoration of the sense of kinesthesia and psychological reassurance are important.

Physical measures employed to aid in the attainment of the objectives listed are thermotherapy, massage, electrical stimulation, muscle re-education, and support. (Proper care of the eye is an important problem, not the province of this discussion.) Kovacs[2] believed strongly that these measures have a decongestive effect and that treatment with them should commence immediately. However, the value of decongestion days after the onset, as mentioned before, is questionable. Nevertheless, it is perfectly rational to begin treatment as early as possible, not only because of the slight possibility that it may decompress the nerve, but because maximum retardation of atrophy[16] is obtained through early and proper therapy. Furthermore, proper therapy, particularly effective electrical stimulation, retards the loss of actomyosin, the contractile substrate of muscle tissue. Fischer[20] has demonstrated that treatment (electrical stimulation) reverses the denervation decrease in the nonexchangeable potassium of muscle.

The authors use infra-red as the thermotherapeutic modality. It is applied to the involved side for 20 to 30 minutes, at least once daily. When pain is present this tends to relieve it and it increases the circulation, particularly the arterial portion.

Massage should follow the application of heat in order to further remove fluids from the tissues. Its effect is greatest on the venous and lymphatic vessels thus completing the stimulation of the circulatory circuit. Furthermore, the massage movements should be such as to mobilize the muscles of the face and thereby help maintain their normal length. If contracture has set in, these same movements may help to ameliorate the condition.

Electrical stimulation is the most important modality but if not administered correctly[16] may be quite useless. It is started early. The current which most effectively causes contraction of muscle is used. In general, the highest frequency which is still optimal for the involved muscle should be utilized. When variable frequency sine wave currents are available then a quick test should soon provide the answer as to the most effective frequency. If there is no reaction of degeneration then a suitable tetanizing current should be used. When there is a reaction of degeneration a sine wave current of low frequency (0.5 to 5.0 cycles per second) is the one of choice. The highest tolerated intensity that does not injure the patient should be applied. Most important is that the muscles being stimulated should be held by the therapist's fingers in order to develop mechanical tension as they contract. The muscles of the face are much like tenotomized muscles and cannot develop tension as they contract unless fixed artificially. The development of tension is essential for maximal results.[22] The duration of one treatment session with electrical stimulation is usually ten minutes. If the contraction response diminishes prior to the ten minutes period the stimulation is stopped since no further value can be expected. However, no harm results if a muscle is stimulated to the point of fatigue.

Another important result of electrical stimulation is the restoration of the sense of kinesthesia. This effect may be seen immediately and consists of the redevelopment of a sense of awareness of muscle function. Frequently, the patient has a highly dramatic subjective and, to a much smaller extent, objective sense of return of muscle function. The examiner can actually see, after the first session, that there are excursions that were not present before the treatment. This is an aid to the program of instruction in muscel re-education.

Electrical stimulation is discontinued as soon as the patient can voluntarily contract unassisted approximately 50 per cent of normal or as soon as the face is nearly symmetrical in the rest position, or at such time as it is decided that no further progress is obtainable.

Muscle re-education is an important part of the therapy; it requires much attention to detailed kinesiology and must be performed by persons well trained in this work. Physical therapists who are registered with the American Registry of Physical Therapy have had this training. Muscle positions and their direction of pull are pointed out to the patient and motion is performed passively and assistively with the patient observing himself in the mirror. When strength develops, carefully graded resistance may be given.

When the cheek or eyebrow droop perceptibly, these tissues may be supported by means of tape fixation.

SUMMARY

Excellent prognosis of facial nerve paralysis, particularly Bell's palsy, has been obtained by means of faradic-galvanic testing and more precisely by electromyography. These tests have their greatest value if performed serially until the third week following the onset when dependence on the findings is unequivocal. It is emphasized that no therapy can be evaluated without prior electrodiagnostic testing because of the wide variety of outcomes, from complete recovery in a few weeks to no recovery ever. Furthermore, the faradic-galvanic test alone is inadequate. It must be combined with electromyographic testing in order to truly evaluate the nerve status. Electromyography of facial muscles is more difficult than of limb muscles and must be performed only by the experienced.

Physical therapy is of value in facial nerve paralysis for its physiological benefits, decongestive effects, its maintenance of muscle tone, and for its retardation of atrophy and retardation of deleterious chemical changes in denervated muscle.

STRITCH SCHOOL OF MEDICINE.

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TABLE 1.
RELIABILITY OF FARADIC-GALVANIC TESTING IN 12 CASES OF BELL'S PALSY FOR PURPOSES OF PROGNOSTICATION.

NO.	PATIENT	SEX	AGE	DURATION OF SYMPTOMS AT TIME OF INITIAL TESTING	R.D.	CLINICAL OUTCOME
1.	PS	M	59	18 days 16 days	None	About 80% recovery noted after 4 weeks Rx. 95% recovery at 11 weeks
2.	JS	M	64	7 days 14 days	Moderately severe partial	About 15% recovery noted 4 weeks after onset. No further follow-up
3.	HA	M	54	30 days	Full (Ramsey Hunt)	30% return of function 17 months after onset
4.	BD	F	64	90 days	Mod. severe partial	No change in function (40%) 6 mo. after onset
5.	MD	M	62	7 days 21 days	None	10% recovery in 3 weeks. Seen 6 months later when he had had complete recovery for some time
6.	EL	F	30	5 days 21 days	None	Complete recovery after 5 weeks
7.	JM	M	64	14 days	None	15% improvement 24 days 30% improvement 31 days
8.	KM	M	48	7 days 14 days	None	20% recovery in 3 weeks upper and lower portions returning first
9.	LM	F	58	7 days 14 days	None	100% recovery in 5 weeks
10.	IR	M		7 days 14 days	None	50% recovery in 3 weeks
11.	AF	F	38	31 days	Mod. partial	90% recovery in 5 months
12.	AM	M	31	17 days	Mild partial	40% recovery in 5 weeks
13	IP	M	25?	14 days	None	15%recovery in 14 days 100% recovery in 4 weeks
14.	IB	M	50?	30 days	Severe partial	40% recovery in 10 months. Mass action
15.	GI	F	48?	8 years	Full	No recovery. Excessive sagging of face

LXXV

EARLY PROSTHETIC CLOSURE FOLLOWING MAXILLARY RESECTION

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The postoperative problem following maxillary resection or palatal antrotomy is often perplexing and frustating to the otolaryngologist or maxillo-facial surgeon, as well as to the patient. To help provide a means of early rehabilitation, the use of a temporary prosthetic device can be very helpful in successful postoperative care of the patient.

The important problems confronting the patient immediately following the removal of the maxilla or part of the palate are the inability to take food, unintelligible speech, and a psychological adjustment to his handicap. The taking of food does not trouble the patient following surgery until the packing is removed from the defect. At this time, the patient upon eating may experience difficulty in chewing or, due to the passage of food into the nasal cavity, sneezing, coughing or regurgitation may result. Due to inarticulate speech, the patient becomes aware of the difficulty of communication. This produces many frustrating experiences in his daily contacts, and often prevents his returning to work. These hardships subsequently lead to psychological stress and periods of depression.

When anticipating the resection of a maxilla and hard palate, the surgeon should discuss with the patient the possible need of a prosthetic device. Coffin[1] believes the patient should be seen one week before surgery and a plastic plate made to fit the hard palate. This should be worn for a few days prior to surgery in order to

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accustom the patient to it, but we feel this is seldom necessary. In considering the patient for a prosthesis, he will usually fall into one of several categories: 1) patient with dentures, either acrylic or similar plastic, 2) patient with a partial denture, 3) patient with own teeth, 4) edentulous patient without dentures. Later we shall discuss the type of prosthesis used in each of these groups.

Although the primary purpose of surgery in such cases is the complete eradication of the tumor or existing pathology, the surgeon, whenever possible, should prepare the soft tissues and adjacent structures for later insertion of a prosthetic device. This can be done by leaving undercuts in the tissues for prosthetic retention; by avoiding crossing the midline of the palate or destroying the septum, except when necessary; by avoiding destruction of the soft palate if it is not involved, since this allows nasopharyngeal control to be retained by the patient. Kazanjian and Converse[2] also advise preliminary surgical measures in simplying the problems of attending prosthetic design, and they feel the main problem in using a prosthesis lies in finding a means of retention.

Our main concern in this paper is the early closure of the maxillopalatal defect by means of a temporary prosthesis. Various temporary methods have been employed but as a whole have proved unsatisfactory. Some of these are: 1) Vaseline and iodoform gauze packs. These produce disagreeable odors, function poorly and need frequent changes. 2) Attempts to fill the defect with various wax substances and dental molding compounds. These have been found to be irritating due to contact with the tissues and the patients frequently complain of their presence. 3) Rubber and natural sponges. These have proved rather unsatisfactory due to inability to be retained in a fixed position, a tendency to acquire an obnoxious odor, and irritation of the soft tissues. 4) Inflatable rubber devices. These are not commonly used today.

We have found early rehabilitation can best be achieved by the use of a temporary prosthesis consisting of a plastic sponge (Ivalon®) implant firmly attached to an acrylic base or to the patient's own denture. This type of sponge is a polyvinyl formalinized substance which is extremely light, resilient, can be easily molded, has qualities of permanence, and most important, is inert to human tissue. Grindley and Clagett[3] used the sponges experimentally in the pleural cavity of several dogs following the removal of a lung. On reopening the chest after several months, they found the sponges to be covered with a thin avascular sheet of fibrous tissue which was adherent to the thoracic wall. In general, the dogs appeared to suffer no deleterious effects. In recent years, these same plastic sponge implants have been

successfully used under the skin of human beings for mammary plasty.

A further advantage of this particular type of sponge is that it is capable of being easily attached to acrylics used in dentures today, or other similar hard impervious plastics. This fact is an invaluable feature in making a temporary prosthesis which aids early restoration of the normal physiologic functions of speaking and eating. An additional advantage, especially to the patient, is the economical aspect.

The exact structural design of each prosthesis will depend on the dental findings of the individual patient. Since malignancy of the maxilla and hard palate occurs most frequently late in life, the majority of patients will have either partial or full dentures. For patients with full dentures, the denture can be of immediate use in the following manner: After the packing is removed postoperatively, the borders of the defect are outlined with methylene blue or a marking pencil. The upper denture is then inserted and immediately removed, the outline of the cavity being visible on the palate contact side of the denture. If the denture is a clear acrylic or plastic, the outline can simply be marked directly on the denture, since the defect can be seen through the transparent substance. The next step is to trim the plastic sponge to the general contour of the cavity, allowing at least one centimeter in excess in all diameters, except superiorly, where the sponge may interfere with bony structures and tend to push the prosthesis downward. A thin paste is then mixed from the acrylic powder (Polymer) and liquid (Monomer) of a cold curing acrylic such as is used in ordinary denture repair. This creamy mixture is applied to the base of the sponge. The base is slightly compressed manually, firmly pressed to the outlined area on the denture, and held in place for approximately fifteen minutes. The sponge is thus firmly affixed to the denture and the resulting prosthesis is ready for immediate insertion. When the lip has been split as in the Mouré and Ferguson incisions, it may be necessary to cut the scar tissue that frequently forms at the frenulum of the upper lip. Alterations for the proper fit of the prosthesis can easily be made by trimming or adjustment of the sponge.

In cases where the patient has a partial denture or at least enough teeth following surgery to hold a partial denture, a prosthetic appliance, retained by clasps on the teeth, may be feasible. However in those cases requiring close observation or irradiation therapy, a piece of softened dental wax can be fashioned to cover the defect and be reproduced in acrylic. This plastic device may be attached

to the sponge in the same manner as described previously in using the full denture.

The edentulous patient who has never worn dentures should also have the simple temporary cover, as just described, and when healing and treatment are complete a permanent prosthesis with teeth can be made.

Although the creation of a temporary prosthesis is basically the problem of the dentist or prosthodonist, the otolaryngologist and maxillo-facial surgeon should be well aware of its merits in tumor surgery. In some cases, the otolaryngologist may have to construct the prosthesis himself, due to lack of dental facilities, or at other times, it may be necessary for him to discuss with the dentist the type of prosthesis needed. It should be remembered that the design, construction and fitting of a good temporary prosthesis is a problem that requires wholehearted effort and cooperation on the part of both the otolaryngologist and the dentist.

During the past six months, we have used a temporary prosthesis in six patients. None has complained of its presence nor has irritation been produced. In most instances, a recording of the patient's voice was made with and without the prosthesis, and a marked improvement was noted after its insertion. An important observation noted was a marked psychological uplift displayed by the patient. This was extremely helpful in contributing to his early rehabilitation. We realize a longer period of observation is necessary and a larger number of cases will have to be analyzed before final conclusions can be reached.

The advantages of using a temporary prosthesis composed of a plastic sponge and acrylic or plastic base are: 1) It affords early restoration of the physiologic functions of speaking and eating within a few days following surgery; 2) the prosthesis can be easily inserted and maintained by the patient; 3) it is relatively inexpensive and simple to construct; 4) this type of prosthesis offers a soft cushion-like surface which does not tend to interfere with healing and with the filling of the defect with granulation tissue; 5) it is readily removable for irradiation therapy and close postoperative observation; 6) the cosmetic appearance of the patient is generally improved by the supporting structure of the soft plastic sponge against the inner surface of the anterior cheek; 7) it tends to promote better hygiene due to the ease of cleansing; 8) its use may be prolonged in those cases where a complete eradication of pathology was not possible at the time of surgery.

SUMMARY

The postoperative problem following maxillary resection or palatal antrotomy is discussed, with suggested measures to obtain early rehabilitation of the patient. A simple, inexpensive temporary prosthesis is described which is composed of a polyvinyl plastic sponge attached to an acrylic or plastic base or to the patient's own dentures. The conditions for using this type of temporary prosthesis and its advantages are presented.

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LXXVI

EFFECT OF VITAMIN K COMPOUND (SYNKAYVITE) ON COMPUTED BLOOD LOSS DURING ADENOTONSILLECTOMY

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The object of this report is to evaluate the effect of vitamin K compound (Synkayvite®) on the amount of blood lost during the performance of adenotonsillectomy. Comparison of blood loss was made between two groups of patients undergoing this operation; one group received this vitamin prior to the operation and the other did not. Not considered here is the effect of vitamin K on bleeding following adenotonsillectomy.[1-5]

METHOD OF COMPUTING BLOOD LOSS DURING SURGERY

A measured quantity of sterile water is used to rinse all the blood from the instruments, and to keep the suction tubing clean during the operation. After hemostasis has been completed, all blood is suctioned from the pharynx and nasal cavity. The measured water is then aspirated through the suction and tubing to flush all contained blood into the suction bottle. The contents of the suction bottle are then measured, the amount of water used is noted, and the actual amount of blood present is derived. The quantity of blood necessary to saturate a tonsil sponge as used by me is approximately two cc. These sponges are counted and the calculated amount of blood is added to arrive at a total figure. Separate suction equipment is used for the aspiration of mucus, saliva and vomitus.

This report presents a series of 176 children, from 2 to 11 years of age, upon whom adenotonsillectomy was performed (1952-1953), and in whom the blood loss was computed in the above manner. To each of 85 cases selected at random, 5 mgm of Synkayvite was administered orally three times daily for one week preceding the operation. The remaining group of 91 cases did not receive this medication. The same operative technique was used in all cases and all pre-operative medication and preparation were the same in both groups except for the vitamin.

In the first group (85 cases receiving Synkayvite) the maximum blood loss in a single case was 150 cc, and the minimum was 20 cc. The average blood loss in this group was 53 cc.

In the second group (91 cases not receiving Synkayvite) the maximum blood loss was 165 cc, and the minimum was 14 cc. The average blood loss in this group was 51 cc.

SUMMARY

The computed blood loss during adenotonsillectomy in a series of 176 children averaged 52 cc. This amount was not significantly affected by the oral pre-operative administration of Synkayvite.

384 Peachtree St.

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LXXVII

SCLEROMA IN EGYPT

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A number of reports have made scleroma well known through the world, but many aspects of the disease require clarification.[1, 2]

The disease is seen not uncommonly now in Egypt, although it is a distinct impression that the incidence of scleroma is decreasing as compared with 20 or 30 years ago. The huge characteristic saddle nose, the involvement of the upper lip, and the perforation of the septum and palate, as described by Madden[4] and Shaheen,[5] is seldom encountered.

Scleroma is a chronic inflammatory disease of the upper respiratory tract, insidious, slowly progressive and benign in nature. It is thought, but not proved, that the etiological agent is *Klebsiella rhinoscleromatis*.[3] As we observe it, the disease attacks almost exclusively the poorer class living under unhygienic conditions.

Because of an unusual opportunity to study several cases, with clinical, histopathological, and bacteriological observations, it appeared that some contribution might be made on the clinical variations observed, the clinico-pathological changes following therapy, with notes on the effectiveness of some newer treatment agents on the disease.

MATERIAL AND METHODS

The 14 cases of scleroma which are reported were out-patients of the Demerdash Hospital, Cairo. Nine were females and five males,

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Collaborative research of the Department of Ear, Nose and Throat, Abbassia College of Medicine, Ein Shams University, with U. S. Naval Medical Research Unit No. 3, Cairo, Egypt.

The opinions and assertions contained herein are the private ones of the authors and are not to be construed as official or reflecting the views of the Navy Department or the naval service at large.

and the age ranged from 16 to 52 years. The patients were all in the lower income group. An attempt was made to classify the cases clinically, and biopsies were taken for histopathological observations, and for correlation with treatment studies. Bacteriological observations were also made. Treatment was then begun with oxytetracycline (Terramycin®), chlortetracycline (Aureomycin®), Streptomycin,® Streptomycin plus isonicotinic acid, or isonicotinic acid alone.

RESULTS

Clinical Description and Classification. Scleroma usually starts in the nose, and progressively involves the nasopharynx, pharynx, larynx and trachea. Occasionally the pharynx is spared. The mucosa and submucosa and muscles are attacked, but bone, cartilage, and lymphatic glands are not affected.

The clinicopathological variations, as observed by us, are as follows:

Stage 1. The Early Catarrhal Stage. Characterized by edema and infiltration with symptoms of discharge and obstruction. This stage is well exemplified by Case 13.

Stage 2. The Atrophic Stage. The mucosa of the nose appears thin, dry, and is covered by small thin crusts, giving the nasal cavity a widened, roomy appearance. Nasal symptoms of dryness and burning, without fetor, are present.

In the pharynx, the mucosa appears glazed with sticky mucous here and there on the surface, with symptoms of dryness of the throat and much clearing of the throat in an attempt to remove the mucous.

In the larynx, the mucosa in the subglottic area looks dry and is covered with thin scales. The rest of the larynx appears normal. A dry cough is usually the only symptom. The atrophic stage is seen either as a primary stage or following treatment. Examples are Cases 11 and 12.

Stage 3. The Granulomatous Stage. In the nose, various appearances are seen:

a) Slightly raised areas of pale grey color due to heaping up of epithelium and slight edema may be seen often next to the vestibule.

b) Discrete granulomas 3-4 mm in diameter and raised 2-3 mm above the mucosal surface may be seen on the septum and turbinates, while the remaining nasal mucosa appears atrophic. These granulomas tend to ulcerate and are usually covered by scabs (Cases 5 and 11).

c) Diffuse granulomatous infiltration of the submucosa, involving any part of the nasal cavity, with scattered ulceration and adherent scabs, removed with difficulty, exposing raw bleeding sites (Cases 2 and 3).

d) Localized nodules, covered with intact epithelium. One case (No. 11) showed a half-spherical nodule 1 cm in diameter anteriorly on the septum.

e) Soft vascular bleeding granuloma. In Case 7, the granuloma appeared to lie on the surface of the mucosa of the lateral nasal wall and soft palate. It was red, soft and bled profusely on touch.

In this granulomatous stage, the internal granulomas encroach on the nasal cavity. Neighboring areas ulcerate and adhere to each other, and to others on opposing surfaces, with the end result of nasal obstruction. Externally the granulomatous masses push against the alae nasi, infiltrate the columella and upper lip, causing deformity. Thus the main complaint of the patient is nasal obstruction. The difficulty of passage of air in the nose, causes anosmia. The lack of ventilation of eustachian tubes leads to varying degrees of tubotympanic catarrh. Among others symptoms are frontal headache, nasal discharge with scabs, slight or profuse bleeding and a nasal vocal tone.

In the pharynx, the diffuse granulomatous type of infiltration is usually found involving the soft palate, uvula, posterior pillars and the lateral parts of the posterior pharyngeal wall. The granuloma involves the mucosa, submucosa and the muscles. As a result of the ulceration, the palate and pillars become matted together and adherent to the posterior pharyngeal wall, closing the nasopharynx from the oropharynx, usually leaving a very small hole of 3-4 mm about the middle line (Case 1). Patients worry little about their throats but usually they complain of slight dysphagia.

In the larynx, the granulomatous changes observed were all subglottic. Case 12 showed discrete granulomas covered with scabs (Stage 3 b), and Cases 4, 6 and 11, showed granulomatous bands below and parallel with the vocal cords, beginning anteriorly below the anterior commissure and fading away posteriorly. The bands were separated from or attached to the vocal cords. There was no ulceration of the bands (Stage 3 c). The patient's complaints are usually cough, dyspnea and stridor, both inspiratory and expiratory, in advanced cases.

In the trachea, variations of the granulomatous stage were seen in Case 12, where discrete granulomatous masses were present in the

lower trachea (Stage 3 b), and in Case 11, where the upper trachea was infiltrated with diffuse granuloma (Stage 3 c). The main symptoms of these patients are productive cough, dyspnea, stridor both inspiratory and expiratory.

Stage 4. The Stage of Fibrosis. The result of irregular change of the granulomatous infiltration into hard contracting fibrous tissue. This results in distortion in the shape of the granulomatous masses and hardness and cartilaginous feeling, causing obstruction of the nose, deformities of the palate, pharynx, and laryngeal-tracheal obstruction.

These clinico-pathological pictures merge into each other and overlap in the whole upper respiratory tract.

BACTERIOLOGICAL ASPECTS

Organisms of the genus *Klebsiella* were first isolated from patients with rhinoscleroma by Von Frisch[6] in 1882. Since the disease could not be reproduced in experimental animals by the inoculation of these organisms, and since the methods available at that time did not permit a distinct differentiation between the rhinoscleroma bacilli and those of the Friedlander-aerobacter group, they were not generally accepted as the etiological agents of the disease.

As late as 1946 it was stated by Topley and Wilson[7] that there was little evidence that these organisms were the causative agents of the disease.

Between 1947 and 1951 Levine, Hoyt and Peterson[8] isolated strains of *K. rhinoscleromatis* from clinical cases of scleroma and presented good evidence of the etiological role of the organisms. They were shown to be biochemically and serologically distinct from other members of the genus *Klebsiella* and were found only in people with scleroma.

Methods: Biopsies and fresh cotton swabs from the nasal mucosa of scleroma patients were received at the laboratory in sterile tubes. The biopsy material was crushed with sterile, rat-toothed forceps and the resulting pulp streaked onto blood, SS (Difco), Mac Conkey (Difco) and eosin methylene blue (Difco) agar plates. The swabs were streaked directly onto the same media.

After 24 hours incubation at 37° C. the plates were examined and nonlactose fermenting colonies transferred to triple-sugar-iron agar (Difco) slants. Strains showing an alkaline reaction toward the lactose slope and acid without gas in the dextrose butt were further tested for their biochemical properties. Those which produced acid

TABLE 1.
ISOLATION OF *K. RHINOSCLEROMATIS* FROM SCLEROMA PATIENTS.

NO.	PATIENT	DATE	RESULT
7	S. A. A. R	12-3-52	Positive
		12-24-52	Positive
		12-30-52	Negative
		1-14-53	Negative
		2-10-53	Negative
		2-24-53	Negative
		3-17-53	Negative
		3-25-53	Negative
		4-14-53	Negative
8	M. A.	12-30-52	Positive
		1-14-53	Positive
		1-20-53	Negative
		2-10-53	Negative
11	B. A.	4-21-53	Positive
		5-5-53	Negative

only in dextrose, maltose and mannitol sugar broths, failed to ferment lactose or sucrose, failed to produce indole and reduced nitrates were forwarded to a typing center for complete serological identification.*

Strains of *K. rhinoscleromatis* isolated during this study were tested for their in vitro sensitivity to the common antibiotics. A freshly transferred strain was thickly streaked on the surface of a blood agar plate. Bacto-sensitivity filter paper discs containing penicillin G, Bacitracin,® Dihydrostreptomycin, Chloromycetin,® Aureomycin, Terramycin and Polymixin® B were placed on the surface of the inoculated plates.

After 24 hours incubation the zones of growth inhibition around the discs were measured.

*Dr. Ida Orskov, State Serum Institute, Copenhagen, Denmark.

TABLE 2.
ZONES OF GROWTH INHIBITION OF ANTIBIOTIC DISCS.

No.	Strain	Penicillin 10 μ	Bacitracin 20 μ	Streptomycin 100 mcg	Chloromycetin 60 mcg	Aureomycin 60 mcg	Terramycin 60 mcg	Polymixin B 30 mcg
7	SAAR 12-30	....	....	8 mm	5 mm (5 mcg)	9 mm	8 mm (10 mcg)	x
7	SAAR 4-8	....	....	x	18 mm	11 mm	21 mm	3 mm
7	SAAR 4-8	....	....	10 mm	19 mm	12 mm	20 mm	3 mm
8	MA 4-8	....	....	x	17 mm	15 mm	16 mm	2 mm
11	BA 4-24	....	....	10 mm	19 mm	20 mm	16 mm	6 mm
11	BA 4-24	....	....	11 mm	18 mm	18 mm	12 mm	6 mm

x Not done.

Results: Biopsy material and nasal swabs from 14 scleroma patients were cultured for the presence of *K. rhinoscleromatis*. The organisms were recovered from three patients (Table 1).

Results of in vitro sensitivity tests are shown in Table 2.

THE HISTOPATHOLOGY OF RHINOSCLEROMA

Szmurlo[8] stated that the first change which takes place is a metaplasia of the epithelium from pseudo-stratified ciliated columnar epithelium to stratified squamous epithelium (Fig. 1). This is followed by a dense cellular infiltration of the submucosa consisting of lymphocytes, plasma cells, polymorphonuclear leucocytes and a few "foam" or Mikulicz cells which appear at the periphery of the infiltrate (Fig. 2). In addition, Russell bodies begin to appear in the infiltrate. These are, eosinophilic, homogenous bodies which are thought to form from degenerating plasma cells (Fig. 3). The development of these bodies can be followed through the various stages from the change in the cytoplasm of the plasma cell from basophilia to eosinophilia to the well-developed hyaline Russell body which measures 20 to 30 micra in diameter. This change is thought to be due to an alteration in the protein of the cytoplasm of the plasma cell. In the early stages the vascularity of the granulomatous lesion is marked and the newly formed vessels are lined by numerous endothelial cells (Fig. 2). At this time fibroblastic proliferation begins and small bands of fibrous connective tissue are laid down within the

infiltrate. The Mikulicz cells are found frequently throughout the dense cellular infiltrate at this stage of development. Degeneration of the Mikulicz cells, plasma cells, and lymphocytes is followed closely by increase in the fibrous connective tissue until the picture consists of a few lymphocytes and Russell bodies scattered throughout a dense fibrous stroma (Figs. 4 and 5). Within the Mikulicz cells there are usually found diplobacilli which are thought to be the bacillus of Frisch or *Klebsiella rhinoscleromatis* (Fig. 6).

In the series of cases presented in this study, biopsies of the nasal lesions were obtained in 7 of the 14 cases. In Case 7 and 8 biopsies were obtained during and after the course of treatment consisting of 100 grams of streptomycin. In the remaining seven cases one or more biopsies were obtained at various times before, during, and after treatment with streptomycin and terramycin. The biopsies were fixed in 10 per cent formalin and processed in the routine manner. Two or three sections of each biopsy were stained with hematoxylin and eosin. Sections from the biopsies were stained with Giemsa stain and Brown and Brenn's stain for bacteria.

TREATMENT

An attempt was made to observe the effect of various antibiotics and drugs, in dosages as follows:

1.	Terramycin	2 gms daily (adult)
2.	Aureomycin	1 gm daily (adult)
3.	Streptomycin	1 gm daily (adult)
4.	Streptomycin + Isonicotinic acid	200 mgm daily
5.	Isonicotinic acid alone	200 mgm daily

The drug, or combination of drugs, was given, if it proved effective, until the case had obtained apparent maximum benefit. By this is meant that all active granulomatous processes had either disappeared or had been replaced by residual fibrosis and all ulcerated areas had become epithelialized. The atrophic state of mucosa and scabbing was found to persist after that and the drug was stopped despite this. Biopsies were taken in Case 7 before the start of therapy and at intervals during the course.

The number of cases reported here are too few to enable us to draw conclusions, but we may offer some observations from treatment of this limited number of cases.

Generally speaking, the disease in its first three stages responded to all the agents used. Rapidity of response depended on several factors:

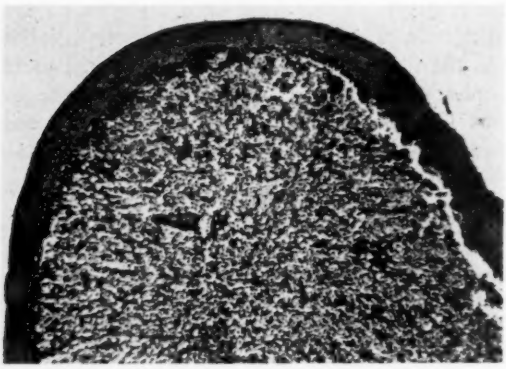

Fig. 1, Case 11.—A nodule of Mikulicz cells and aggregates of plasma cells covered by hyperplastic stratified squamous epithelium. H & E x 160.

1. Stage of the disease.

Case 13, in the early catarrhal stage (Stage 1), and all cases in diffuse granulomatous type of the third stage (Stage 3 c) were the quickest to respond. Cases in the second atrophic stage (Stage 2) responded only in the sense that a nasal swab previously positive became negative within six or seven days; but the atrophic state of the mucosa, roominess of the nose and scabbing persisted. The localized nodular type of the third stage (Stage 3 d) was most refractory to therapy (See Cases 3, 4, 6, 11).

2. Site of affection.

Generally speaking nasal cases were quicker to respond to antibiotics. Laryngeal cases showed some relative resistance to antibiotics.

3. Kind of antibiotic or drug used.

Though the number of cases treated by each drug individually is not enough for detailed comparison, it appears to us that Aureomycin is the most effective of those used. It did not fail to effect a clinical cure in any of the cases in which it was given.

Streptomycin was also effective in all the cases in which it was given but on the whole it required a longer course.

The response to Terramycin was somewhat irregular. Thus, while in all nasal cases in which it was applied it achieved a clinical cure with a rapidity definitely superior to Streptomycin and com-

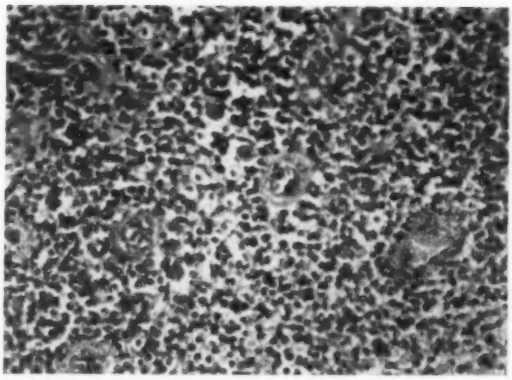

Fig. 2, Case 7.—After 4 grams of Streptomycin. Subacute granulomatous infiltrate composed Mikulicz cells, many plasma cells; numerous thicked wall vessels are present. H & E x 400.

parable with that of Aureomycin, some laryngeal cases appeared to be partially or totally unaffected by the drug. Isonicotinic acid alone had no effect at all on the disease. But combined with Streptomycin it appeared to enhance the action of the latter. Regular observation of these cases after stopping the drug for three months did not show any clinical relapse.

Three points may be made concerning therapy:

1. Antibiotic therapy appears most effective in the first three stages of the disease.

2. Antibiotics appear valueless in the fourth fibrotic stage of the disease.

3. Antibiotics appear of undoubted value in some laryngeal and upper tracheal cases, carrying the patient over a critical period, and saving him from a tracheotomy which would otherwise be essential.

REPORT OF CASES

Case 1.—(N. M. I.) Female 16 years of age, single. Complained of nasal obstruction. Previous history: four years complaint of nasal discharge, scabbing and obstruction. Masses were removed surgically from the nose, followed by deep x-ray therapy, but obstruction persisted and progressed to become complete after three

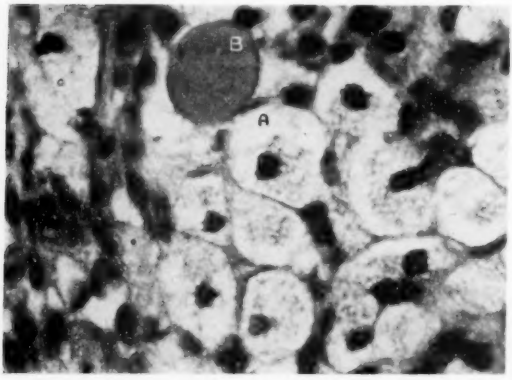

Fig. 3, Case 7.—Higher power of Mikulicz cells (a) and a degenera-ating plasma cell of Russell body (b). H & E x 800.

months. One year ago developed hoarseness, accompanied by a gradually developing obstruction. Hospitalized with acute laryngeal obstruction necessitating urgent tracheotomy. Clinical diagnosis: Scleroma of nose, pharynx and larynx. Wassermann reaction negative. Biopsy (from pharynx): "Only a small area of the piece examined is covered by epithelium, the rest is ulcerated. Moderate amount of fibrosis and a moderate number of Mikulicz cells and Russell bodies. Blood vessels are fairly thick walled. The appearance is that of a scleroma passing towards the fibrosing stage." (Stage 4). X-ray of chest was clear. X-ray of larynx showed irregular subglottic narrowing characteristic of scleroma.

Streptomycin injections and deep x-ray therapy were given. The appearance after two months was as follows:

Nose: Fibrous adhesions in both anterior nares. No granuloma, scabs or fetor. No external deformity or tenderness. Scar tissue completely closed the nostrils and was covered by glistening epithelium.

Pharynx: Fibrosis of soft palate, which had become nearly immobile and adherent to the posterior pharyngeal wall on both sides, leaving a small central hole of communication (with the nasopharynx) through which purulent discharge issued.

Larynx: A cresentic subglottic scar. No active granuloma. No obstruction. After antibiotics for one week, the fibrous tissue

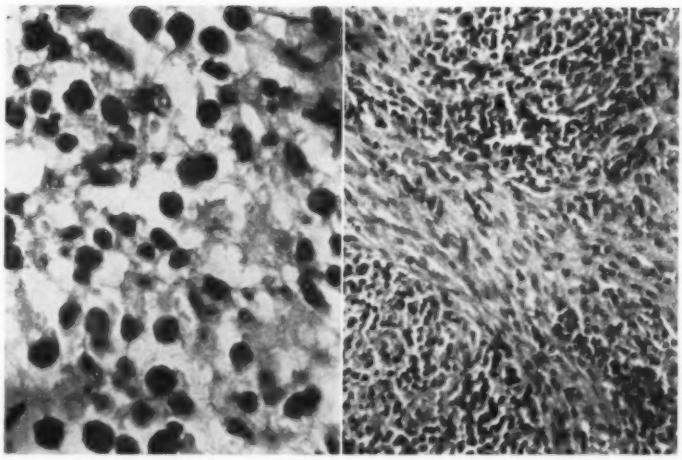

Fig. 4, Case 7.—After 30 grams of Streptomycin. Dense fibrous strands penetrating the granulomatous infiltrates. H & E x 800.

Fig. 5, Case 7.—After 30 grams of Streptomycin. Degeneration of the Mikulicz cells and diminution in the number of plasma cells. H & E x 800.

in the anterior nares was excised completely. The nasal fossae behind were found free. Skin graft in each nostril was successful. Two weeks later the soft palate was freed from the posterior pharyngeal wall on both sides. Naso-pharynx was skin grafted. Tracheotomy tube removed and tracheotomy wound was allowed to close.

Comment: The scleroma in this case is of the fourth stage of fibrosis. It is peculiar that in this case which was treated by deep x-ray therapy, no scabbing was present, unlike cases treated with antibiotics.

Case 2.—(S. A. S.) Female, 18 years of age. Complained of nasal obstruction, tenderness, swelling and deformity of nose, and epistaxis of nine months' duration. No history of trauma or syphilis.

On examination there was widening of the dorsum of the nose below the bony part, with a definite bulge on the left side, cartilaginous to the touch, and tender on palpation. Anterior rhinoscopy showed nasal passages to be narrowed due to diffuse infiltration by granulomatous process on both sides of septum and on anterior ends

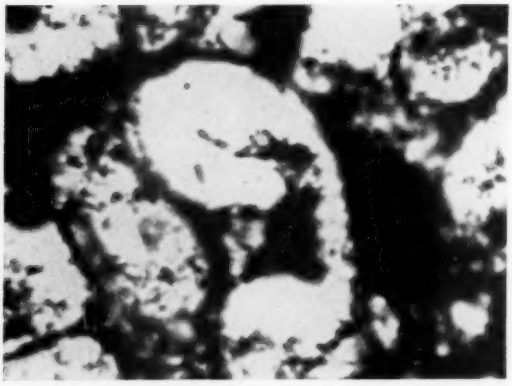

Fig. 6, Case 11.—Bacilli of Frisch within a Mikulicz cell. Brown and Brenn's stain. x 1000.

of both inferior turbinates (Stage 3 c). Posterior rhinoscopy was negative. Pharynx and larynx were normal.

Clinical diagnosis: Rhinoscleroma. Wassermann reaction negative. Biopsy: "The microscopic picture is that of a rhinoscleroma. It is partly covered with stratified squamous epithelium which shows evidence of hyperplasia in some areas. There are numerous Mikulicz cells and Russell bodies present. There is a fair amount of fibrosis. The blood vessels show no abnormality."

Terramycin (2 gms daily) was begun. After 22 days of treatment examination showed marked improvement in the width of the nasal passages, with diminution of infiltration and a tendency to atrophic appearance of the mucosa in some areas. Biopsy at this stage still showed the full histological appearance of rhinoscleroma. After 120 gms of Terramycin the condition was considered clinically cured. Three months later, no clinical relapse was seen. Biopsy showed a granulomatous process with few Mikulicz cells containing Diplobacilli.

Comment: This patient showed the clinical and characteristic histopathology of rhinoscleroma of the diffuse granulomatous infiltrative type (type 3 c). She received 120 gms of Terramycin after which the clinical appearance was normal. The histological picture of scleroma, however, persisted even after three months from the end of treatment. No clinical relapse was seen after three months.

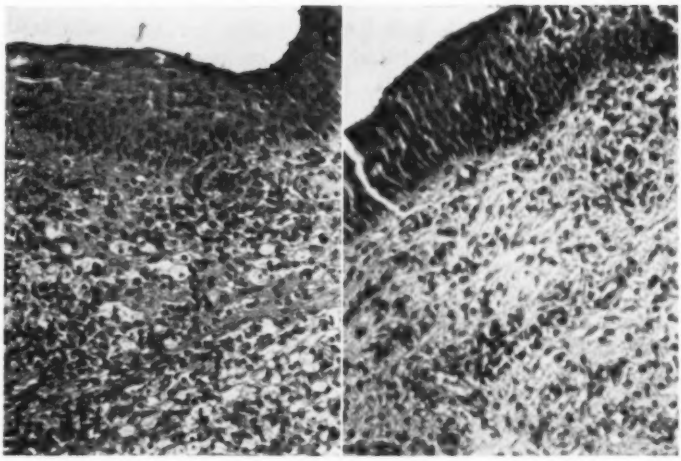

Fig. 7, Case 7.—After completion of 100 grams of streptomycin. Granulomatous infiltrate persists although it has diminished and is being replaced by strands of fibrous tissue. H & E x 400.

Fig. 8, Case 7.—47 days after completion of streptomycin. Scattered aggregates of lymphocytes within a fibrous tissue stroma. H & E x 400.

CASE 3.—(M. A. A. M.) Male, aged 52 years. Complained of nasal obstruction with discharge and scab formation in the nose, occasional epistaxis and dryness of the throat, of six months' duration. No history of syphilis.

Examination showed no external deformity of the nose. No tenderness. Anterior rhinoscopy showed granulomatous infiltration on both lateral and septal walls of both fossae, more proliferative in discrete areas with areas of less production between (Stage 3 c). The nasal passage were markedly encroached upon. Few scabs were present and on their removal the underlying areas were found to be granulating with no intact surface epithelium. There was scanty bleeding on removing the scabs. Posterior rhinoscopy showed a pale bluish mass on the left side of the posterior end of the septum covered with intact mucosa (Stage 3 d). To the touch this mass was found to be soft. Pharynx and larynx were free.

Clinical diagnosis: Rhinoscleroma. Wassermann reaction negative.

Biopsy: "The appearance is that of rhinoscleroma. The granuloma is partially covered with stratified squamous epithelium. There are many Mikulicz cells but Russell bodies are not encountered. Fibrosis is minimal and the blood vessels are normal." Biopsy from the posterior mass showed also the histological picture of rhinocleroma.

Terramycin (2 gms daily) was given for 30 days, with marked improvement in the width of the nasal passages. The granulomatous process diminished but there were still areas of residual infiltration which became refractory to treatment. Scabs were also present. The posterior mass was not affected at all. The treatment was changed to streptomycin 1 gm daily. Clinical improvement was evident after 60 days; anterior rhinoscopy showed almost complete disappearance of the granulomatous process. Scabbing was still present. The posterior mass diminished to about half of its size.

Comment: This case of rhinoscleroma was partially refractory to Terramycin. The use of Streptomycin resulted in improvement. Scabbing persisted after clinical disappearance of the granulomatous process. The histopathology was that of diffuse granulomatous infiltration (Stage 3 c). The mass on the posterior end of the septum was of the localized nodular type (Stage 3 d). It was noticed that the nodular type was more refractory to treatment.

Case 4.—(S. A. Y.) Male aged 25. Complained of hoarseness, sensation of foreign body in the laynx, respiratory difficulty and cough, slowly progressive for nine months. No history of syphilis or tuberculosis.

Examination showed no external deformity of the nose, fetor, or tenderness. Anterior rhinoscopy: Roomy nasal passages, atrophic mucosa, scanty discharge with scabs. Posterior rhinocopy negative. Pharynx was free. Larynx showed the characteristic clinical appearance of laryngoscleroma (type 3 c). Subglottic stenosis caused by pale fleshy infiltration in the subglottic area on both sides below the vocal cords starting anteriorly but stopping short of the interarytenoid region. The cords did not appear to be infiltrated and were freely mobile. No surface ulceration was detected but scabbing was seen. The amount of cough and respiratory distress was found on repeated clinical examination to vary in proportion with the amount of scabs.

Clinical diagnosis: Laryngoscleroma. Wassermann reaction negative. Sputum negative for tubercle bacilli. X-ray of larynx and chest: Subglottic stenosis characteristic of laryngoscleroma. Chest clear.

Terramycin (2 gms daily) was begun. Respiratory distress, hoarseness, and cough improved. Scabbing of nose improved, but did not disappear.

After receiving 40 gms no further improvement was noticed. Treatment was continued. The patient began to complain again of respiratory distress and cough. The subglottic infiltration appeared to be fleshy and active. Terramycin therapy was stopped after 66 gms and the patient was put on Streptomycin 1 gm daily. There was definite response, judging from the amelioration of symptoms and clinical appearance. After 80 gms of Streptomycin the fleshy subglottic infiltration was completely replaced by a fibrous band simulating a web. Treatment was stopped. Re-examination six months later showed no recurrence.

Comment: This case of laryngoscleroma failed to respond to Terramycin. The symptoms and clinical appearance after some improvement became worse while the patient was still under treatment. A good clinical response was obtained by Streptomycin after failure of Terramycin.

Case 5.—(G. A. E.) Male 18 years of age. Complained of gradual and progressive nasal obstruction for three months. No history of syphilis.

Examination: Nose—septum deviated to the right. Patchy infiltration of both lateral and medial walls of both nasal fossae. Areas of infiltration are not much raised on the surface and show a granular appearance with surface ulceration and scabs (Stage 3 b). The areas of mucosa not affected are atrophic. The right side is more heavily involved. No adhesions and no fetor. Pharynx and larynx were free.

Clinical diagnosis: Rhinisclerома. Wasserman reaction negative. Biopsy showed typical appearance of rhinoscleroma.

Terramycin 2 gms daily was begun. Repeated examination showed gradual regression of the infiltrative process. After the administration of 40 gms, the granulations disappeared completely, nasal obstruction was relieved, and the patient was considered as clinically much improved. Scabs did not disappear, however.

Comment: This case shows that patients of this type (Stage 3 b) are amenable to antibiotics; 20 days' treatment was sufficient to achieve good clinical improvement.

Case 6.—(M. A. M.) Female 25 years of age. Complained of acute laryngeal obstruction, progressive for four months. Cough

and hoarseness appeared later, with repeated epistaxis. Syphilis was denied. Patient pregnant, nearly full term. Nose—no external deformity or tenderness. Roomy nasal passages. Atrophic condition of mucosa. Scanty discharge with scabbing (Stage 2). Pharynx—a localized nodule was found on the middle of the margin of the left posterior pillar (Stage 3 d). Nasopharynx was free. Larynx—subglottic area almost completely occluded granuloma below the cords on both sides. The chink left was covered by discharge and dried scabs (Stage 3 c).

Clinical diagnosis: Scleroma. Wassermann negative. Tracheotomy performed. Biopsy from nodule on posterior pillar: "This is a scleroma covered with stratified squamous epithelium. It is partly proliferative, with numerous Mikulicz cells. Russell bodies are scanty and there is a moderate amount of fibrosis." From larynx: "This is a scleroma which is only partially covered with squamous epithelium. There are very few Mikulicz cells. Russell bodies are absent. Fibrosis is only scanty. Vessels are normal." X-ray of chest was clear. X-ray of larynx: Subglottic stenosis compatible with a laryngoscleroma.

Streptomycin 1 gm daily was begun. She received a total of 130 gms with the following results: Subglottic infiltration was replaced by a crescentic scar. Nodule removed from left posterior pillar did not recur. Atrophic condition of nasal mucosa was the same. Tracheotomy tube was removed and wound allowed to close.

Comment: The patient delivered three days after admission, before the start of treatment. Delivery was followed by marked regression of subglottic proliferation. Response to Streptomycin was considered good.

Case 7.—(S. A. A. R.) Female, aged 25. Complained of severe nasal obstruction and severe epistaxis, progressive for four years.

The nose showed a reddish, non-ulcerated, fleshy, polypoid mass in the right nares, extending from the lateral wall to the floor (Stage 3 e). The mass bled easily on touch, and profusely on biopsy. In the left nares, a similar mass, fleshy, reddish, granulomatous (Stage 3 e) caused adhesion between the lateral wall and septum. No external deformity or fetor. The pharynx showed extension of the fleshy, polypoid, vascular mass over the nasal surface of the soft palate, which was not ulcerated, but bled easily on trauma (Stage 3 e). It appeared to lie on the surface of the palate without infiltrating it. Palatine movements were normal. Biopsy from the nasal lesion showed "atrophic squamous epithelium intact, with a granulomatous infiltrate composed of numerous 'foam' or Mikulicz cells,

plasma cells and small aggregates of polymorphonuclear leucocytes. Many new formed vessels, lined by swollen endothelial cells, were scattered throughout the infiltrate. Numerous Russell bodies, which appeared a spherical eosinophilic inclusions, from 1-20 micra in diameter, were seen, and appeared to be formed from degenerating plasma cells. Focal areas of edema were present around the vessels. No fibrosis was seen. Giemsa and Brown and Brenn's stains showed numerous diplobacilli within the Mikulicz cells (Figs. 2 and 3)." Material from this biopsy showed, with special bacteriological methods, a positive culture for *K. rhinoscleromatis.*

Treatment with Streptomycin, 1.0 gm daily was continued for 100 days. Improvement took place, with complete disappearance of the granuloma in the nose and pharynx. The left nares, however, showed fibrous adhesions between the inferior turbinate, the floor, and the septum, which showed no change up to 45 days following the end of treatment.

A nasal biopsy on the 30th day of treatment showed "moderately large areas of fibrosis within the granulomatous infiltration and focal areas of degenerating Mikulicz and plasma cells (Figs. 4 and 5)."

A nasal biopsy five days after completion of treatment showed "granulomatous infiltrate consisting predominently of plasma cells, with a few Mikulicz cells scattered throughout. Numerous diplobacilli were present within the Mikulicz cells (Fig. 7)."

A nasal biopsy on the 45th day after treatment showed "an intact respiratory epithelium, with submucosa composed primarily of young loose, fibrous tissue with several small aggregates of lymphocytes, a few macrophages and eosinophiles. Only a few plasma cells were present. No Mikulicz cells or Russell bodies were noted (Fig. 8). No organisms were present in the sections stained with Giemsa or Brown and Brenn's stain." Comment: This patient showed nasal and pharyngeal scleroma in a polypoid granulomatous stage (Stage 3 e) with nasal respiratory obstruction. The clinical diagnosis was confirmed by histopathological and bacteriologic techniques.

Treatment with Streptomycin was followed by clinical disappearance of granuloma, with residual intranasal scarring. Biopsies showed progressive change from subacute granuloma to a chronic type of granuloma with fibroblastic changes, and finally the disappearance of the typical histopathological evidence of rhinoscleroma.

CASE 8.—(M. A.) Male 27 years of age. Complained of nasal obstruction and thick discharge with scab formation; slowly progressive for three years. Syphilis was denied. Examination: Nose —No external deformity or tenderness. Anterior rhinoscopy showed narrowing of the nasal passages. Mucous membrane of the septal and lateral walls on both sides showed diffuse granulomatous infiltration. Surface ulcerated in several areas and covered with scabs (Stage 3 c). Granulations were firm to touch and gave rise to scanty bleeding. Pharynx, larynx and ears were free.

Clinical diagnosis: Rhinoscleroma. Wassermann reaction negative.

Biopsy: "This is a rhinosclercma showing the following microscopical features: Covering epithelium is stratified squamous. There are plenty of Mikulicz cells, and a fair number of Russell bodies. Fibrosis is not much. Blood vessels are normal."

Terramycin 2 gms daily, was begun, and marked clinical improvement attained after 44 days treatment. Scabbing persisted.

During the course of treatment serial biopsies were taken from the nose every ten days. The results are as follows:

No. 18/53: "A small area only is covered by epithelium. There are numerous Mikulicz cells and Russell bodies. There is definite fibrosis. Vesels show no important change."

No. 43/53: "Covering epithelium is thinned out a little. Number of Mikulicz cells is less than previous specimen. Russell bodies are present but not numerous. Fibrosis is the same. Blood vessels are thick walled."

No. 76/53: "The granuloma is covered by epithelium. The cellular appearance is not much changed, but there is marked fibrosis, and blood vessels are thick walled."

Nasal swabs were taken from this patient serially for bacteriological examination for rhinoscleroma bacillus. Results were as follows:

on 13-12-52	positive
on 14-1-53	positive
on 20-1-53	negative
on 10-2-53	negative

After 42 grams of Terramycin the biopsy was as follows: "A chronic granuloma containing many plasma cells, numerous Mikulicz

cells, many Russell bodies, and a fair amount of fibrosis. Special staining revealed no intra- or extracellular organisms." Two weeks after completion of treatment the histopathology was the same. Biopsy 14 weeks after completion of treatment showed: "Several nodules of degenerated plasma cells, moderate fibrosis, Mikulicz cells, and Russell bodies absent."

Comment: This case (Stage 3 c) improved with Terramycin therapy. It is also noticed that unlike cases treated with radiotherapy (e. g. Case 1), scabbing persisted after the end of treatment. Fibrosis was also less marked and no adhesions were left. The microscopical picture at the end of treatment was that of a non-specific granuloma.

Case 9.—(A. G. M.) Male 12 years of age. Complained of nasal obstruction, thick nasal discharge with scabs and occasional epistaxis, slowly progressive for six months. No history of syphilis.

Examination: Nose—externally normal. Anterior rhinoscopy showed narrowing of nasal passages. Granulations covered with scabs were scattered all over the mucosa on both sides (Stage 3 b). Scabs were continuous almost forming casts of the nasal fossae. Fetor not noticeable. Posterior rhinoscopy negative. Pharynx, larynx and ears were free.

Clinical diagnosis: Rhinoscleroma. Wassermann reaction negative.

Biopsy: "Piece examined is covered by stratified squamous epithelium. The subepithelial tissues are the site of a chronic granulomatous type of inflammation. There are few Mikulicz cells and focal areas of fibrosis. Special stains show Diplobacilli resembling Frisch Bacillus inside Mikulicz cells." Nasal swab was negative for rhinoscleroma bacillus. Terramycin 1 gm daily was begun, with gradual disappearance of granulations and ulceration. After 50 gms, only scabbing persisted. For three months after the treatment the condition was stationary. Biopsy after the end of treatment showed: "Loose fibrous tissue containing scattered aggregations of plasma and mononuclear cells. No Mikulicz cells."

Comment: This case (Stage 3 b), showed the rapid response to antibiotics and the attainment of clinical improvement after a short course, without relapse. Scabbing persisted. The residual pathological picture was that of a nonspecific granuloma.

Case 10.—(W. I.) Female 20 years of age. Complained of complete nasal obstruction, sensation of dryness in the nose and occasional epistaxis for four years. No history of syphilis.

Examination: Nose—no external deformity, tenderness, or change of feeling. Anterior rhinoscopy showed atrophic mucosa and discrete small granulations scattered on both lateral and medial walls on both sides (Stage 3 b), covered by dry scabs. On removal of scabs there were few ulcerations and scanty bleeding. No fetor. Posterior rhinoscopy was negative. Pharynx, larynx and ears were free.

Clinical diagnosis: Rhinoscleroma. Wassermann reaction negative.

Biopsy: "The lesion is a rhinoscleroma. It is partly covered by stratified squamous epithelium. It is a granulomatous lesion in which there are numerous large vaculated Mikulicz cells and a fair number of Russell bodies. There is a moderate degree of fibrosis. It is vascular but the blood vessels are thin walled. Special stains showed Diplobacilli resembling Frisch Bacillus in the Mikulicz cells." Biopsy material showed no rhinoscleroma bacillus on culture.

Streptomycin 1 gm daily was begun. After four weeks granulations almost disappeared but scabs persisted. Treatment was stopped after administration of 30 gms and case taken as clinically arrested. Clinical examination one month from end of treatment showed no change. Nasal biopsy 56 days after arrest of treatment showed a "dense plasma cell infiltration with moderate fibrosis. No Mikulicz cells were present. Many plasma cells showed marked degeneration."

Comment: This case (Stage 3 b), improved on Streptomycin therapy. As in previous similar cases there is persistence of scabs, absence of recurrence and a residual picture of a nonspecific granuloma.

Case 11—(B. A.) Female 50 years of age. Complained of nasal obstruction, difficult respiration and hoarseness progressing slowly for five years.

History began eight years ago when the patient first complained of gradually developing respiratory difficulty and hoarseness. There was nasal discharge which was thick and contained scabs. Diagnosed as scleroma and took x-ray therapy. This made the obstruction acute necessitating an urgent tracheotomy. One year later she could dispense with the cannula. One month before admission, she began to develop symptoms of obstruction and hoarseness again.

Examination: Nose—Atrophic condition of mucosa. Some areas covered with scabs, under which there was discrete multiple masses of granulations. Localized nodular mass on the left side of

the nasal septum covered with intact mucosa (Stage 3 d). No adhesions. No fetor. No external deformity, tenderness or change of consistency (Stage 3 b). Pharynx—Atrophic condition of mucosa (Stage 2). Larynx—Stenosis of subglottic are especially anteriorly. Arytenoids edematous. Cords mobile but edges covered with scabs (Stage 3 c).

Clinical diagnosis: Relapsing rhinolaryngoscleroma. Wassermann reaction negative. X-ray of larynx—Subglottic stenosis compatible with scleroma. X-ray of chest negative. Smear positive for *K. rhinoscleromatis* on 4-21-43. Smear negative on 5-5-53. Biopsy (from larynx): "The appearance is that of a cellular rhinoscleroma. Surface layers of covering epithelium are degenerated, thin, and partly ulcerated. Thick strands of fibrous tissue are seen near the surface in one area and penetrating into sub-epithelial layers. There are other thin branching bands of fibrous tissue here and there. Mikulicz cells are very numerous and Russell bodies present, but to a less extent. Blood vessels are numerous in cellular areas but few and thick walled in area of fibrosis."

Aureomycin 2 gms daily was given for one week, with marked improvement in the symptoms and clinical picture. Gastric disturbances necessitated reduction of the dose to one gram daily. After two weeks the appearance was as follows: Larynx: Granuloma in subglottic area replaced by a thin crescentic scar. Nose: Localized nodular mass unaffected. Mass removed surgically. It proved to be rhinoscleroma and it did not return.

Comment: This case shows the relative resistance of the localized nodular type of Stage 3 d in the nose to antibiotics (see also case 3). Aureomycin appears more rapid in action on laryngoscleroma than other antibiotics.

Case 12.—(F. A.) Female 30 years of age. Complained of nasal obstruction with discharge and attacks of epistaxis; also hoarseness and attacks of dyspnea and dryness of throat. Onset of symptoms insidious and course slowly progressive. Syphilis denied.

Examination: Nose—No external deformity, tenderness or change of feeling. Anterior rhinoscopy showed narrowing of nasal passages; mucosa atrophic and partly covered with scabs; removal of scabs reveals small granulomatous areas (Stage 3 b). Pharynx—Mucous membrane dry, glazed and atrophic (Stage 2). Larynx—Mucosa, pale, thin, glazed and atrophic. Thick sticky secretion with scabs in the glottis. No ulcerations and subglottic area is free (Stage 2). Trachea—Discrete granulomatous masses at the lower end of trachea (Stage 3 b).

Clinical diagnosis: Rhinoscleroma. Wassermann reaction negative.

Biopsy: "A cellular rhinoscleroma. Only a small area is covered by stratified squamous epithelium. This is degenerated and markedly thinned out. There is a moderate degree of fibrosis seen especially towards the superficial layers of the lesion. Thin strands are also seen scattered throughout. Mikulicz cells are relatively few, Russell bodies present. Blood vessels tend to be thick walled." X-ray of larynx and chest negative.

Isonicotinic acid 50 mgm, six hourly, was begun. After two weeks there was no improvement. A combination of isonicotinic acid and Streptomycin (1 gm daily) was given with improvement, and in three weeks the patient was discharged.

Comment: Isonicotinic acid alone proved to be of on effect. Combined with Streptomycin the effect of the latter appeared to be enhanced.

CASE 13.—(A. A. M.) Female 19 years of age. Complained of nasal obstruction, purulent nasal discharge with scab formation in the nose, and anosmia.

Onset indefinite, and course slowly progressive. No history of syphilis.

Examination: Nose—Slight tenderness on pressure externally, but no change of feeling or external deformity. Anterior rhinoscopy showed the mucosa to be thick and oedematous. This scabs present on scattered areas and surface underneath raw and granular. Thick tenacious discharge. No adhesions, but the air passage is markedly encroached upon (Stage 1). No fetor. Pharynx, larynx and ears negative.

Clinical diagnosis: Rhinoscleroma. Wassermann reaction negative.

Biopsy: "Covering epithelium is one layer columnar in areas and shows in other areas transition into low squamous. On the whole it is edematous and partly degenerated. There are some glands in the sub-epithelial layer, one of which is distended into a cyst like structure. There is a fair number of Mikulicz cells, but Russell bodies are totally absent. Vascularity is not a feature."

Terramycin 2 gms daily, was begun. Gradual improvement was observed. The granulating raw surfaces disappeared. Thickened mucosa became reduced with improvement in the width of the

nasal air passages. Scabbing persisted. After the administration of 44 gms the case was considered as clinically arrested. No change was noted on repeated observation for one and one-half months following cessation of treatment.

Comment: This case was first catarrhal or invasive stage. Vacuolated cells of Mikulicz were present while Russell bodies were absent. The case responded well to Terramycin therapy in a relatively short course.

CASE 14.—(O. M. M.) Female, aged 20 years, complained of nasal obstruction and discharge, and gradual and progressive. No history of syphilis.

Examination: Nose—Swelling below the nasal bones causing external deformity. The bulge was more marked on the left and was cartilaginous to the touch. Anterior rhinoscopy showed diffuse thickening of both lateral and medial walls of both nasal fossae, with deflection of septum to the right. Nasal passage reduced to a minimum (lateral and medial walls on the right came together in several places) (Stages 3 c). Pharynx, larynx and ears were negative.

Clinical diagnosis: Rhinoscleroma. Wassermann reaction negative.

Biopsy: A typical appearance of rhinoscleroma.

Terramycin 2 gms was given daily for 22 days, after which she was considered clinically arrested. Further observation for three months showed no recurrence.

Submucous resection of the deflected septum was done later. A piece of nasal septal cartilage with mucosa on one side was sent for biopsy, which showed: "a nonspecific fibrosing reaction involving the perichondrium and subepithelial layers; the cartilage was normal."

Comment: This case demonstrates that the disease does not appear to attack the cartilage. A good response was obtained with Terramycin.

COMMENT

The differential clinical and histopathologic diagnosis of well developed rhinoscleroma is not difficult. The earlier lesions are suggestive of atrophic rhinitis and the later stage may be easily confused with syphilis. The histopathology may be confused with that of leprosy, when the Mikulicz cells are prevalent. The foam cells of leprosy and the Mikulicz cells are very similar; however, they can

be differentiated by the presence of diplobacilli or the bacillus of Frisch in the latter. In the 14 cases presented here the initial biopsies were characteristic of rhinoscleroma. Mikulicz cells, plasma cells, Russell bodies and varying degrees of fibrosis were found in each. In each the mucosa consisted of stratified squamous epithelium, which indicates a metaplasia from the typical pseudo-stratified ciliated epithelium found in the respiratory area of the nose.

Streit[1] stated that Mikulicz cells may develop from various cells as fibroblasts, plasma cells, histiocytes, leucocytes and epithelial cells. No evidence of development of the Mikulicz cells and fibroblasts, plasma cells, leucocytes or epithelial cells was present in the histopathological material studied in this series. It is probable that the Mikulicz cell represents a macrophage or reticulo-endothelial cell which has phagocytized the bacilli of Frisch or *Klebsiella rhinoscleromatis*. According to Forbus'[9] concept of certain types of granulomatous diseases the macrophages or reticulo-endothelial cells may serve as essential sites of growth and propagation of the causative agents. The viral granulomatous diseases such as lymphopathia venereum, fungus diseases, such as coccidiomycosis and histoplasmosis, as well as some of the bacterial diseases, such as leprosy and typhoid fever are examples of this. Furthermore, the intracellular position of these viruses, fungi, and bacteria may be the primary factor in producing the proliferation of the parasitized macrophages. This process may well be applied to rhinoscleroma.

It is considered, from the results in the cases presented, that the histopathology of the lesion after treatment is a valuable adjunct to the clinical evaluation of the response to treatment.

SUMMARY

1. Fourteen cases of scleroma as seen in Cairo, Egypt are presented, with a proposed classification of the disease.

2. Histopathological and bacteriological observations are discussed.

3. Results of treatment with newer antibiotic agents are discussed.

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NAMRU-3, c/o American Embassy, Cairo, Egypt.

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The Scientific Papers of the American Broncho-Esoghagological Association

LXXVIII

TRACHEOGRAPHIC AND BRONCHOGRAPHIC STUDIES AS AIDS IN THE DIAGNOSIS OF CONGENITAL MALFORMATIONS OF THE TRACHEOBRONCHIAL TREE AND AORTIC ARCH IN INFANTS AND CHILDREN

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At the Fourth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology, the authors presented an exhibit on "Bronchography in Infants and Children." This exhibit was well received and prompted us to record some of our ideas and experiences in the diagnosis of congenital lesions of the tracheo-bronchial tree and aortic arch, which we believe to be far more common than is generally supposed. We shall try in this brief discussion to show how the instillation of a contrast medium into the lower respiratory tract of certain patients, together with endoscopic procedures, may be of great assistance in the graphic demonstration of hitherto unsuspected, and often obscure, conditions. A few of the cases we are demonstrating here have been previously reported as single case histories, or included in papers on a special subject, from the clinics at The Children's Medical Center, Boston.

Two years ago, at the Toronto meeting of this organization, Holinger (et al)[1] presented an excellent and comprehensive paper on many such anomalies based on Jordan's classification. Several other articles along similar lines may be obtained from the extensive bibliography accompanying Holinger's paper in the Transactions of the American Broncho-Esophagological Association.

TECHNIQUE

In our bronchoscopic work we prefer general anesthesia for most infants and all children, consisting ordinarily of premedication with

From The Children's Medical Center, Boston, Mass.

Nembutal® and atropine followed by drop ether. Frequently, instead of Nembutal, rectal Avertin® by rectum in doses of 80 mgm per kg of body weight, is used as a basal anesthetic, supplemented by ether. In our hands, with a capable department of anesthesiology accustomed to long and difficult operations on the smallest infants, general anesthesia has given us most satisfactory results over a period of nearly two decades. We find that such methods are quite safe and involve no appreciably increased risk of any kind to the patient. Such procedures leave the patient without apprehension or psychological trauma, which are important considerations when future or prolonged hospitalizations or surgery are contemplated.

A complete bronchoscopic examination is performed with the aspiration of bronchial secretions for bacteriological cultures, sensitivity tests, guinea-pig innoculation, or other studies, and this is followed by the instillation of iodized oil through a flexible endotracheal tube inserted after the bronchoscopic procedure has been completed. With the tube in place, laryngeal spasm can no longer obstruct the airway should the patient become light under the anesthetic. Should the anesthetic state become too profound, oxygen may easily be administered by the positive pressure method. Aspiration of the oil (after the radiographs are made) can be rapidly carried out as completely as possible with no further instrumentation. Filling of the areas to be outlined is done under fluoroscopic guidance via a polyethylene catheter passed through the intubation tube. The patient may then be turned in various positions for adequate filling of all lobes and as many films made as the situation requires. The oil is then aspirated as completely as possible with the same polyethylene catheter, and the intubation tube removed. The amount of iodized oil used obviously depends upon the size of the patient and the condition of his tracheobronchial tree at the time the procedure is performed. The amount varies from 1 to 2 cc for a tracheogram in the newborn to 15 to 20 cc for older children with bronchiectasis. In many instances, in small infants where we wish to demonstrate the type of tracheal compression present (without the necessity of filling the bronchial tree), we carry out a simple rapid procedure using no anesthesia, to obtain an outline of the trachea only. Through an anterior commissure laryngoscope, 1 to 2 cc of iodized oil is instilled directly into the subglottic region through a small polyethylene catheter. The patient has been previously placed on a cassette, and the technician's finger is already on the button. An instantaneous lateral view, (the most important one) is then made, followed immediately by one single anterior-posterior film. The oil is then rapidly aspirated. This usually gives good visualization of the trachea, and may satisfactorily confirm the diagnosis of a suspected vascular ring

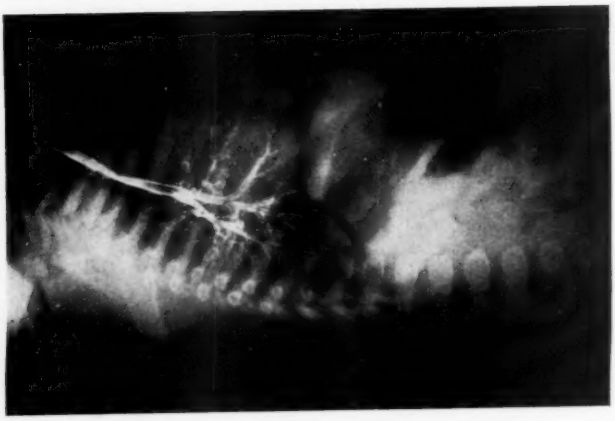

Fig. 2.—Narrowing of trachea due to partial glottic obstruction (lateral view). Seen only during phase of inspiration.

Fig. 1.—Congenital stenosis of trachea (lateral view). Note similarity to Fig. 2.

Fig. 3.—Deficiency of tracheal cartilage (lateral view). View of a serial tracheogram on a three month old infant showing variation in caliber of trachea.

Fig. 4.—Deficiency of tracheal cartilage (lateral view). View of a serial tracheogram on a three month old infant showing variation in caliber of trachea.

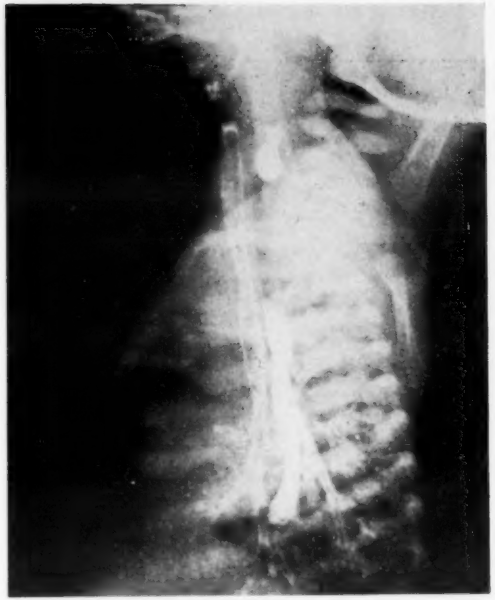

Fig. 5.—Deficiency of tracheal cartilage (lateral view). View of a serial tracheogram on a three month old infant showing variation in caliber of trachea.

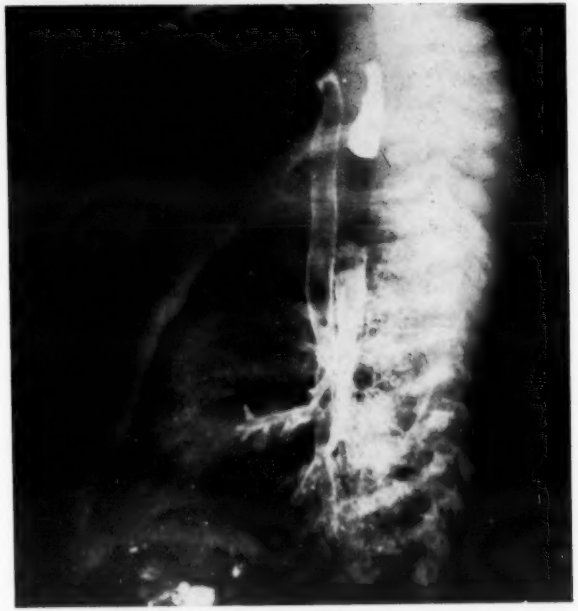

Fig. 6.—Same patient postoperatively showing marked improvement in airway. (Maximum caliber of trachea in a series of 18 films.)

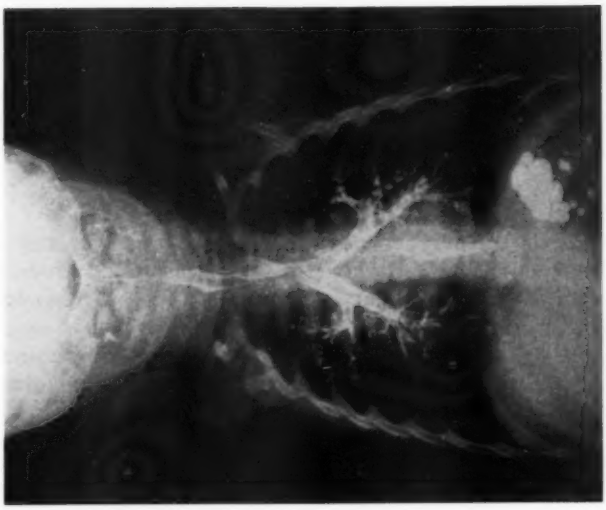

Fig. 8.—AP view of six month old infant showing double aortic arch. Note constriction of trachea and esophagus at same level.

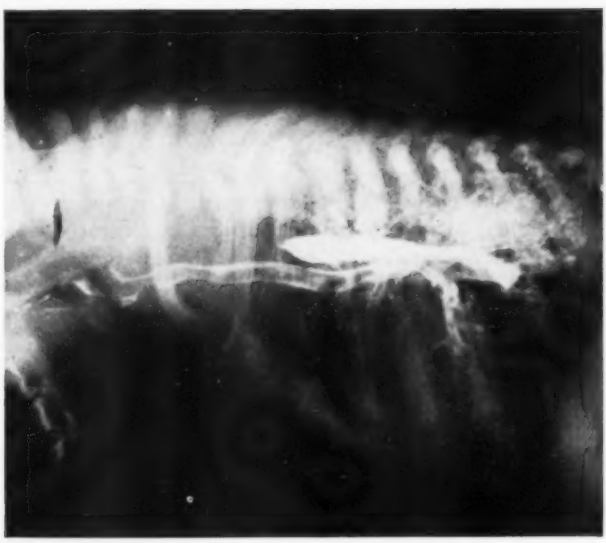

Fig. 7.—Same patient postoperatively showing marked improvement in airway. (Minimum caliber of trachea in a series of 16 films.)

or some other type of compression. If, however, this simple procedure does not satisfactorily demonstrate the suspected malformation, direct endoscopic visualization is always indicated, often with a serial tracheogram. In our clinic, we ordinarily carry out the complete endoscopic examination in practically all patients. Needless to state, a successful undertaking always presupposes teamwork of a high order on the part of the anethesiologist, the radiologist, and the endoscopist.

TRACHEAL STENOSIS

Occasionally a film is obtained which, from the gross appearance, suggests tracheal stenosis, either localized or generalized. Such patients usually exhibit stridor along with other signs of respiratory obstruction. Figure 1 shows stenosis of the trachea, a true organic stricture, through which it was not possible to pass a 3 mm bronchoscope. Figure 2 shows a similar trachea, but in the latter instance there is an apparent narrowing seen only during inspiration and disappearing during expiration. Here, the narrowing is physiologic and results from partial glottic obstruction. Such narrowing will disappear when the glottis is held open with the laryngoscope, and the tracheal lumen will remain normal throughout the respiratory cycle. Such a picture of apparent stenosis may be due to anomalies of the epiglottis, redundant aryepiglottic folds in an exaggerated infantile larynx with an omega-shaped epiglottis, redundant mucosal folds on the arytenoids, to the arytenoids themselves, or, possibly, to a congenital tumor of the larynx. The apparent stenosis appears during inspiration and is paradoxical. It is physiologic rather than anatomic.

In patients with tracheomalacia, no symptoms are noted while the bronchoscope is in place beyond the defect, nor is any difficulty noted in passing the bronchoscope downward. On withdrawal of the tube, the patient exhibits a resumption of the inspiratory or expiratory stridor (many have both), dyspnea, and, sometimes, cyanosis, due to the flaccid, collapsible trachea. Serial tracheograms may help to demonstrate graphically such conditions, showing all gradations from a severe obstruction at one time to an almost normal lumen seconds later. A three-months-old infant who had severe inspiratory stridor, dyspnea, retraction and cyanotic attacks was reported to have a normal larynx. However, on examination of the trachea the midportion showed marked variation in caliber with an apparent localized defect of the cartilaginous framework. A serial tracheogram showed marked variation in the size of the lumen as noted in Figures 3, 4 and 5. Doctor Robert E. Gross conceived and performed a procedure in which the thymus was removed, the great

vessels and aortic arch were pulled forward with sutures and along with the adventitial tissue of the trachea in its defective portion, stitched to the posterior aspect of the sternum. Shortly after the operation the trachea appeared as noted in Figures 6 and 7. These are two views from a postoperative serial tracheogram series showing the maximum and minimum lumen, with approximately a fifty per cent improvement in the airway, which resulted in marked improvement in the patient's symptoms. This procedure has been carried out in other patients having similar findings with the same gratifying results. Our experience with these patients has demonstrated to us that the cause of stridor and retraction sometimes will be found lower down in the trachea, and not solely in the larynx as is commonly supposed. Unless the trachea is carefully studied in all patients with stridor or obstruction not satisfactorily explained by laryngeal findings, the source of the symptoms will continue to go unrecognized. In these patients general anesthesia has made careful observation and x-ray studies much simpler.

ANOMALIES OF THE AORTIC ARCH AND GREAT VESSELS

About ten years ago, Doctor Gross[2] in the course of his pioneer work in cardiovascular surgery, became interested in infants with severe external compression of the trachea and esophagus. Anomalies of the aortic arch had always been recognized at the autopsy table, but rarely clinically, and had been regarded as not amenable to surgical correction. In 1945 Doctor Gross performed the original operation, which he had devised, at The Children's Medical Center in Boston on a patient with a vascular ring encircling the trachea and esophagus. We had demonstrated the ring endoscopically and by x-ray studies. Now nearly one hundred and fifty patients with anomalies of the aortic arch and great vessels have been diagnosed and operated upon at The Children's Medical Center, with often dramatic results and a surprisingly low mortality.

DOUBLE AORTIC ARCH

The normal human has an aortic arch derived from the fourth left embryonic arch which crosses the trachea anteriorly, courses to its left, and proceeds downward as the descending aorta. The fourth right embryonic arch disappears in the normal development of the fetus. In patients with a true vascular ring, both embryonic arches persist, the ascending aorta bifurcates around the trachea and usually the esophagus, the arches then rejoining to form the descending aorta. In such patients the anterior (or left) arch is usually the smaller, even though under ordinary circumstances, the anterior should have been the normal one. In most instances a double aortic arch pro-

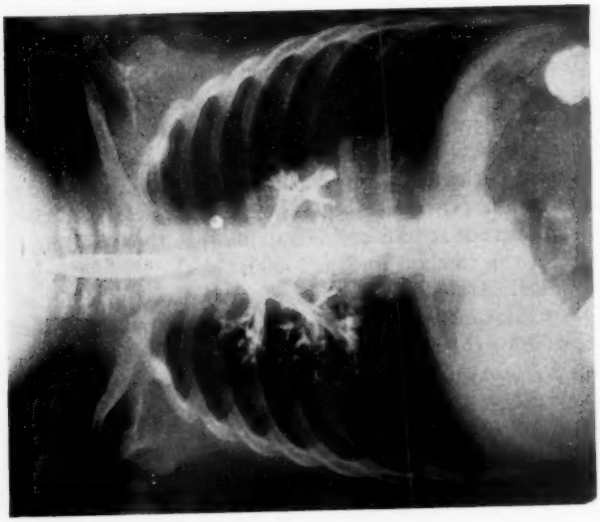

Fig. 10.—AP view of trachea of one year old child showing right aortic arch with left ligamentum arteriosum.

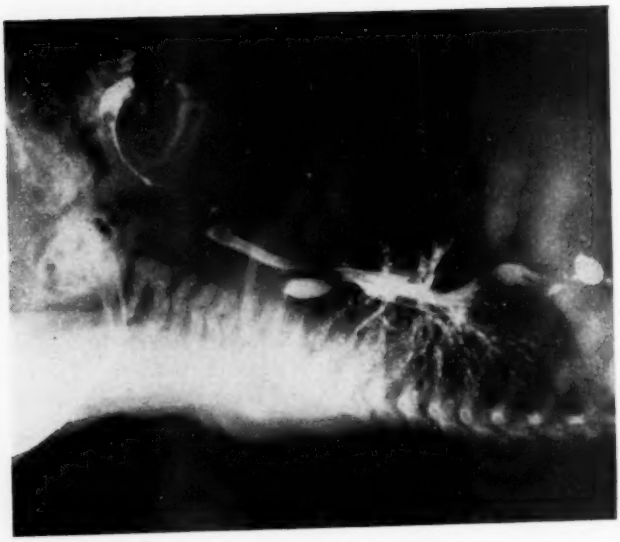

Fig. 9.—Lateral view of same patient with double constriction at 3rd and fourth thoracic vertebrae.

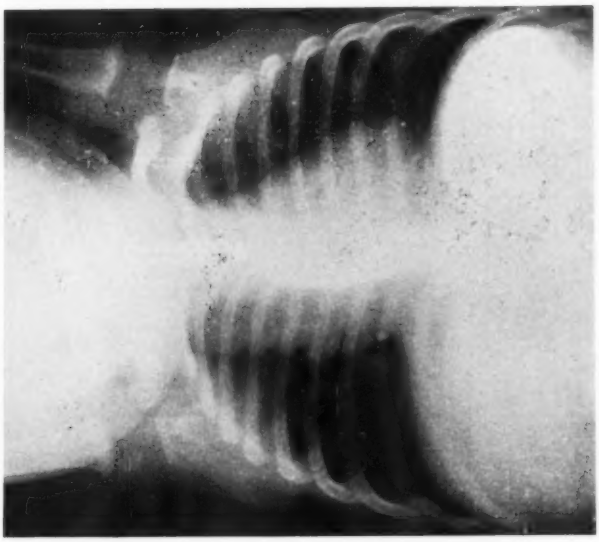

Fig. 12.—AP view of esophagus of same patient showing bilateral constriction.

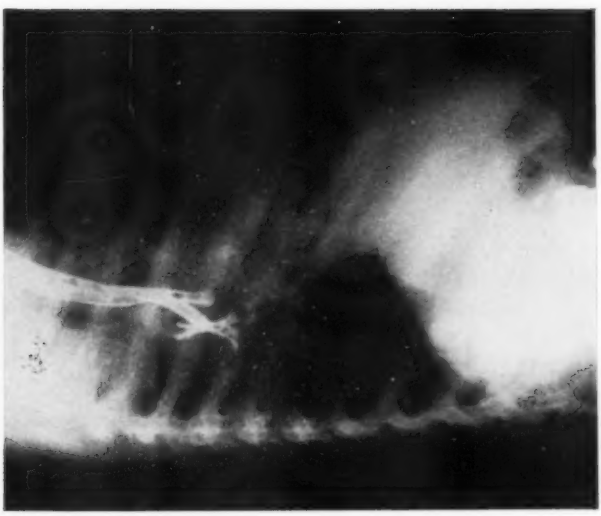

Fig. 11.—Lateral view of trachea in same patient showing compression.

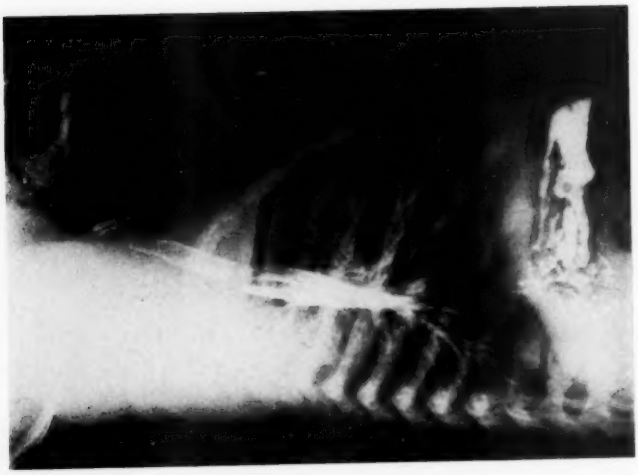

Fig. 14.—Lateral view of trachea of patient with anterior compression from aberrant innominate artery.

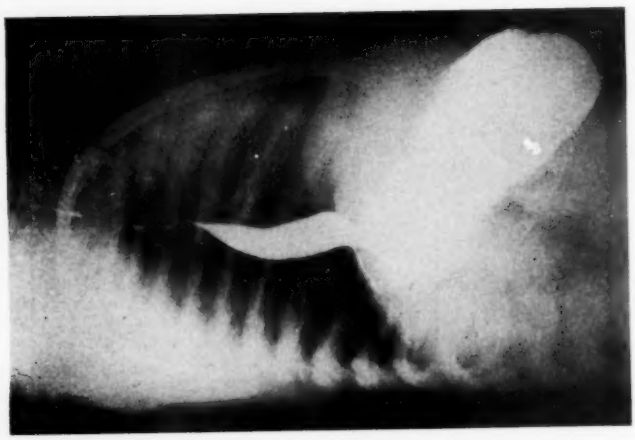

Fig. 13.—Lateral view of esophagus in same patient showing marked esophageal constriction.

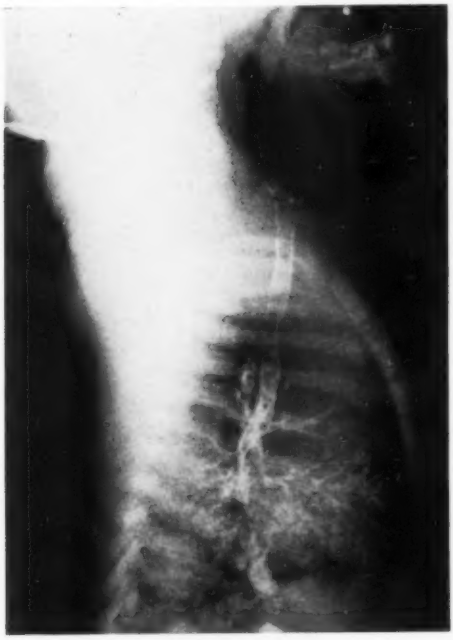

Fig. 15.—Lateral view of trachea of patient with anterior compression from aberrant left common carotid artery.

ducing a vascular ring will give symptoms of a distressing character before the age of six months. Almost always there is some difficulty in swallowing which may vary from a slight hesitation and prolonged holding of food in the mouth to severe dysphagia. The tracheal compression produces severe respiratory embarrassment, with increased respiratory rate, dyspnea, wheezing, retractions, periodic attacks of cyanosis, and inspiratory as well as expiratory stridor. The patient usually lies on his side with the head and neck hyperextended and breathes with a noisy rattle. Pneumonitis frequently recurs and the lungs are often hypoventilated. On bronchoscopic examination one finds a constriction of the lower portion of the trachea, with some forward displacement. The constriction is in the anterior-posterior diameter (Fig. 8). The esophagus shows a similar posterior indentation at the level of the third and fourth thoracic vertebrae (Fig. 9). In the absence of a mediastinal mass, this picture is pathognomonic of some type of vascular ring. Operation consists in ligating

Fig. 16.—Tracheo-esophageal fistula without atresia of esophagus. Note "tenting" effect on anterior wall at location of the poorly filled fistula.

and dividing the smaller of the two arches (usually the anterior) which is preferably done between the left common carotid and the left subclavian artery. After removal of the thymus to relieve further compression, the common carotid is stitched to the posterior surface of the sternum. Division of the posterior limb of the arch (where this is the smaller) allows the arch to move anteriorly in the mediatinum leaving the situation anatomically normal and usually relieving any further constriction.

RIGHT AORTIC ARCH WITH LEFT LIGAMENTUM ARTERIOSUM

In instances where the left fourth embryonic arch disappears instead of the right, the main arterial channel (right aortic arch)

courses to the right and posterior to the trachea and esophagus, continuing downward as the descending aorta, either to the right or the left of the vertebral column. In many patients this anomaly will cause no symptoms of constriction. However, the ligamentum arteriosum must of necessity connect the pulmonary artery anteriorly with the first portion of the descending aorta posteriorly, and if the arch is on the right, the ligamentum must curve around the left side of the trachea and esophagus, and posterior to it in most instances, to reach its destination in the right-sided aorta. This completes a constricting ring, consisting of a right arch and left ligamentum. Such a condition may not give any symptoms if the ligamentum is long or lax, but it may produce severe distress if the ligamentum is short or tense. The symptoms and signs are similar to those seen in patients with double aortic arch but usually are not severe until twelve to eighteen months of age, about a year later than the symptoms bringing patients with double aortic arch to operation. Roentgenograms show the arch to the right of the trachea with a prominent mediastinal shadow, a slight but rather longer indentation of the right tracheal wall, with the anterior compression from a tightly bound pulmonary artery (Figs. 10 and 11). The ligamentum may cause a sharper depression on the left of the trachea. The esophagus is constricted at the same level (third or fourth thoracic vertebrae) on the left side and posteriorly, possibly a bit lower down than the double arch, and with a little narrower and deeper constriction (Figs. 12 and 13). Doctor Gross operated upon the first patient with this anomaly in 1948. The operation is relatively simple, and consists in breaking the constricting ring by severing the ligamentum and allowing the pulmonary artery to fall forward away from the trachea, the trachea and esophagus dropping backward and to the left. If the left subclavian artery runs posterior to the esophagus, as it well may with a right arch, it is ligated and severed. Although the arch remains on the right, the constricting ring has now been relieved. In both of these anomalies it is important to recognize the condition early, so that further compression of the already severely deformed tracheal cartilages will be prevented, and the trachea allowed to develop as best it may. Unfortunately the rings may always continue to show some deformity.

ANOMALOUS INNOMINATE OR LEFT COMMON CAROTID ARTERY

Two other anomalous conditions of the arch and great vessels have been recognized and treated successfully by surgery. These are much more rare and the symptoms much less striking than in the previously described conditions. In both instances there is no difficulty in swallowing, and the esophagus is found to be normal

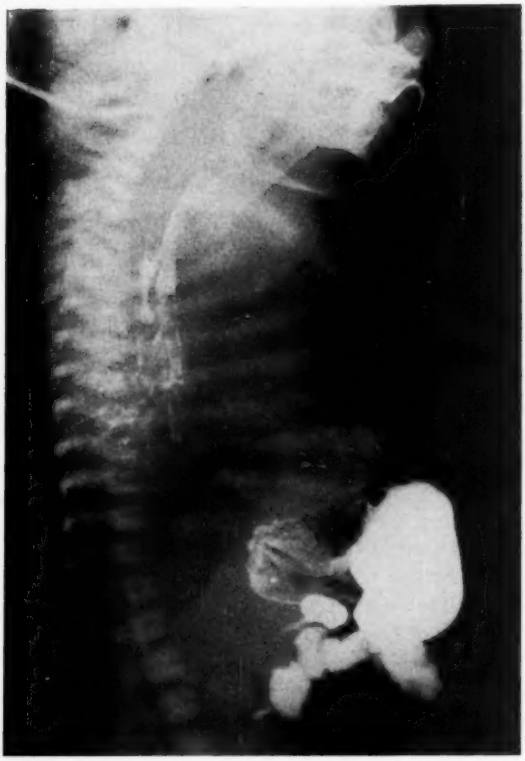

Fig. 17.—Tracheo-esophageal fistula without atresia of esophagus (5 day old infant) lateral view.

both by endoscopic and x-ray examination. However, there is usually inspiratory and expiratory stridor, retraction, dyspnea, rapid respiration, and sometimes cyanosis. Pneumonitis has also been observed frequently in these patients. In order to relieve the compression of the trachea, the patient usually holds the head in marked hyperextension. In one patient we have seen, the airway could be completely occluded by merely flexing the neck. Endoscopically, one may see an anterior tracheal compression which can be demonstrated by lipiodol studies. If the innominate artery arises too far to the left on the aortic arch, it must cross the trachea anteriorly in a line running obliquely upward from left to right to reach the right

side of the neck (Fig. 14). Conversely, if the left common carotid artery arises too far to the right on the arch the compression will run upward from right to left (Fig. 15). In the absence of any abnormal mediastinal shadows suggesting a cyst or tumor such an anomaly of the arch is the most likely diagnosis. Such patients may be confused with those having maldevelopment of the tracheal cartilages (tracheomalacia, etc.) Serial tracheograms will show that in these latter patients the tracheal lumen diminishes considerably during the expiratory phase of the respiratory cycle; whereas, if there is compression from without, the lumen is more constant and the defect unchanging. Operation in either type of anomaly consists in dislocating the abnormally placed vessel anteriorly after removal of the thymus and suturing its adventitial coat to the posterior surface of the sternum thereby relieving the compression. It is very difficult to distinguish these two anomalies pre-operatively; but if the symptoms warrant, exploration is indicated in any event, and the operative approach is identical. Angiograms are therefore not deemed necessary in such a situation.

ABERRANT RIGHT SUBCLAVIAN ARTERY

The aberrant right subclavian artery does not properly belong in this discussion, since the trachea or bronchi are not involved, and there are no respiratory symptoms. However, it is a closely related condition. In these patients one finds a spiral defect of the posterior esophageal wall, producing the clinical picture of "dysphagia lusoria," recognized since the 18th Century. It is caused by an independent origin of the right subclavian artery from the descending aorta rather than from the inominate artery where it normally arises. In its course to the right apex and the right arm, it may severely compress the posterior esophageal wall, if the aorta is in its usual left position. This vessel can be safely severed, because of the excellent collateral circulation of the second and third portions.[2]

TRACHEO-ESOPHAGEAL FISTULA WITHOUT ATRESIA OF THE ESOPHAGUS

The dramatic story of esophageal atresia and its associated fistula is now well known to this group. Prior to 1939 not a single case had been successfully operated upon, while at present the survival rate following surgery is better than 75 per cent. Only in recent years has it become apparent that fistula without atresia is not uncommon. Symptoms are not ordinarily present at birth and often the patient goes through many attacks of pneumonitis before the diagnosis is even suspected. Three years ago one of us[3] reported before the Triological Society a series of five cases of fistula without

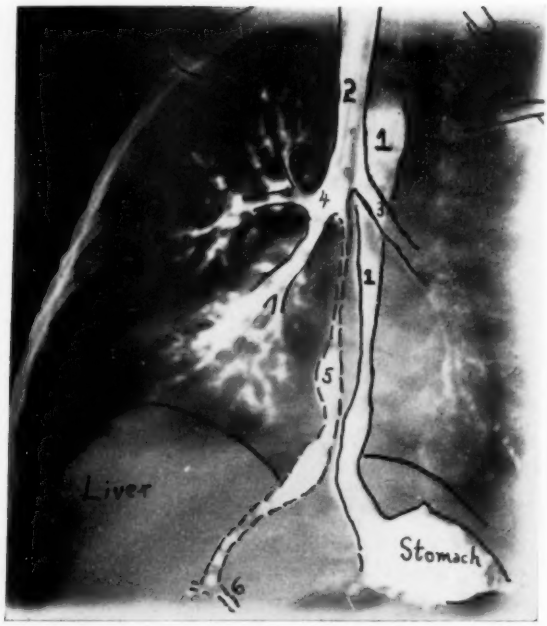

Fig. 18.—Broncho-cholodochal fistula in 5 months old baby: 1. esophagus; 2. trachea; 3. l. main bronchus; 4. rt. main bronchus; 5. fistulous tract; 6. hepatic duct.

esophageal atresia two of which had been successfully operated upon by the end of the second month of life and are still living normal lives. Operation must be carried out before irreversible pulmonary changes have taken place. Early diagnosis is therefore essential. Symptoms are rarely overt, so that any vague respiratory or digestive complaints in early life should arouse suspicion. Frequent attacks of pneumonitis of the aspiration type are the rule, but often there may be only a persistent loose cough compatible with chronic bronchitis. If the fistula is small the infant may eat, gain weight and develop properly. However, attacks of choking with or without cyanosis may occur, and are usually associated with the ingestion of fluids rather than solid foods as the latter do not go through the fistula so readily. Such attacks are most apparent after the infant has finished feeding and is placed in the customary prone position. Simple regurgitation carries the material through the fistula. In such patients there is an increase in frothy mucus, distension is a

Fig. 19.—AP bronchogram. Show congenital stenosis of right lower lobe bronchus with resultant bronchiectasis.

problem, and radiographs almost always show more than the normal amount of air in the gastrointestinal tract. Atresia of the esophagus with fistula should be suspected on the first attempt at feeding and one may rule out an atresia simply by the passage of a catheter. Unfortunately there is no easy method of diagnosis of fistula where no atresia of the esophagus exists. On occasion it may be possible to demontrate a large fistula by the swallowing of Lipiodol® (not barium), but the infant should be placed in the prone position immediately after and several spot films taken rapidly. Even then only one film may indicate the fistula, and it may be incompletely outlined. Frequently only a suggestion of "tenting" of the esophageal wall is seen (Fig. 16). Dyes have been used, but without notable success. Endoscopically, such fistulae are very difficult to visualize, even with the Broyles telescope. Figure 17 demonstrates a lateral view of such a fistula in a five-day-old infant.

Fig. 20.—Agenesis of right lung (age 4 months). AP view. Note marked distortion of tracheobronchial tree.

BRONCHO-CHOLODOCHAL FISTULA

In 1949 we encountered a most unusual case of fistula which was reported by our radiologist (et al).[4] A five months old, poorly developed and nourished, marasmic infant weighing only 7 pounds 13 ounces was admitted after several previous hospitalizations elsewhere. Since birth she had constant respiratory difficulty, associated with the coughing up of large amounts of brownish yellow mucus, which was not actually regurgitated. There had been several attacks of pneumonitis associated with fluctuating areas of atelectasis. On bronchoscopic examination we were able to demonstrate a fistulous opening in the medial wall of the right main bronchus just below the carina. Injection of this fistula with lipiodol showed a definite tract running parallel to the esophagus extending into the region of the liver (Fig. 18). It was possible to inject 30 cc of the oil without producing any increase in respiratory distress. Under the fluoroscope the tract seemed to communicate with the ampulla of Vater. It was not possible to improve the patient's extremely poor general

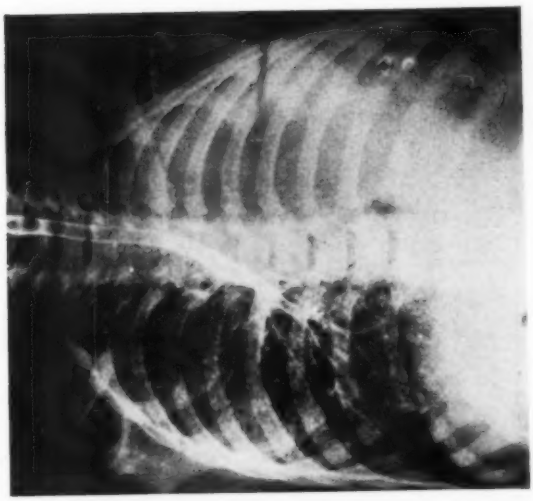

Fig. 22.—Agenesis of left lung (age 3 years). AP view. No sign of left bronchial tree.

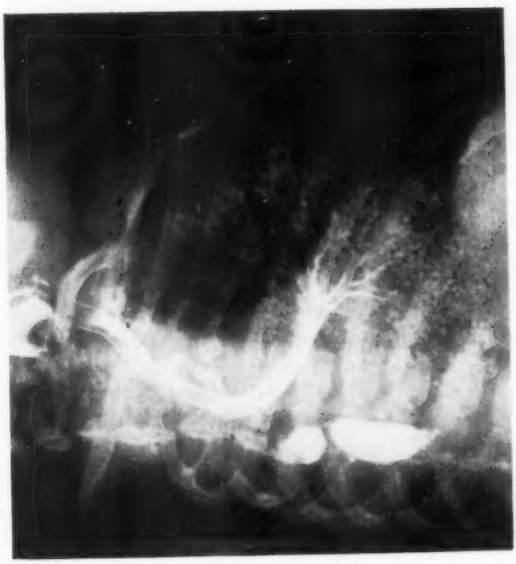

Fig. 21.—Agenesis of right lung (same patient). Oblique view.

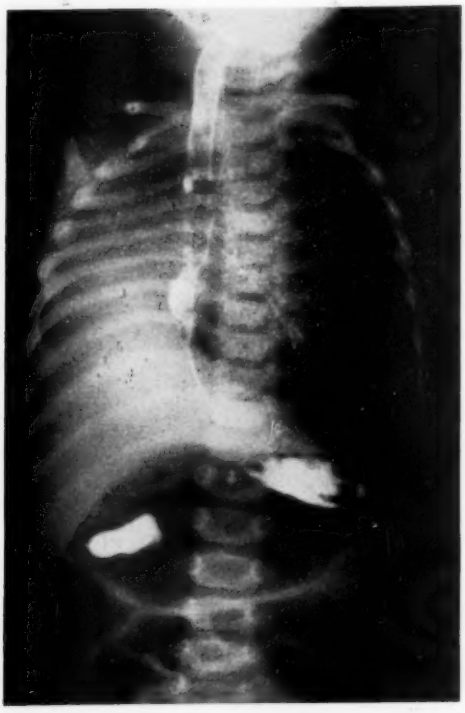

Fig. 23.—Agenesis of right lung (age 4 months). AP view. Note bronchial "bud" and over-expansion of left lung.

condition to withstand exploratory operation and she subsequently died. Post-mortem examination showed a fistulous tract 0.7 cm in diameter coursing down the mediastinum just to the right of the esophagus, entering the abdomen through the esophageal hiatus and passing into the left lobe of the liver. It then became continuous with the hepatic duct, emptied into the common bile duct, and thence into the duodenum. Microscopic examination was interesting in that the upper 2 cm of the tract showed a histologic picture of respiratory epithelium, possibly laryngeal or tracheal, with stratified squamous non-cornified epithelium surrounded by irregular cartilaginous masses. The central portion of the tract was typical of the histology of the esophagus, with a muscularis mucosae and two well developed muscle layers. The lower part of the tract was lined with pseudo-

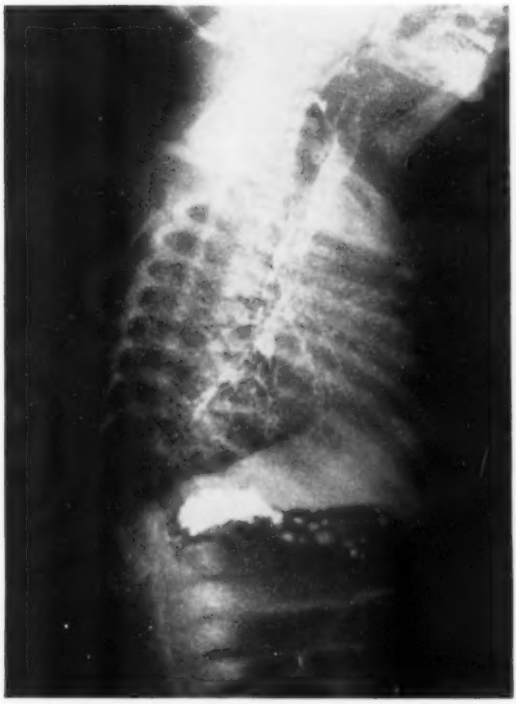

Fig. 24.—Agenesis of right lung (same patient). Lateral view.

stratified columnar epithelium, compatible with the histologic appearance of the biliary system. It was thought the tract might represent a reduplication of the upper gastro-intestinal tract, but this supposition would not explain the varied mucosal pictures and the cartilaginous structures in its upper portion. Dr. J. Lewis Bremer, Consulting Embryologist to The Children's Medical Center, suggested the possibility of a fusion of an anomalous bronchial bud with an anomalous bile duct anlage at the very primitive 4 to 7 mm stage. Had the former extended caudally and the latter cephalad, the two could have remained in apposition and fused, later canalizing into a single tract.

BRONCHIAL STENOSIS

Occasionally an intrinsic obstruction in the wall of a bronchus may be the cause of recurrent pneumonitis or atelectasis, producing

symptoms of cough, wheezing, noisy respirations and later leading to very extensive bronchiectasis (Fig. 19). Localized defects in the bronchial cartilages, analagous to those seen in the trachea, such as absent or defective rings may give the clinical and roentgenographic picture of obstructive emphysema, indistinguishable from that due to a foreign body. During inspiration air will enter the affected lung, but becomes entrapped during expiration just as effectively as in the ball-valve action produced by a peanut fragment. This is easily demonstrated endoscopically as well, by the flapping or fluttering of the bronchial wall in this area, concomitant with the transmitted noise.[5] Subnumerary or supernumerary bronchi are seen very commonly in routine endoscopic work in infants and children, often without related symptoms. On the other hand there may be intermittent wheezing, recurrent pneumonitis in the affected area, emphysema or atelectasis. A tracheal bronchus to an accessory lobe, or to a normal lobe, is not unusual, most commonly arising from the right side of the trachea just above the carina. According to Bremer[6] a tracheal bronchus is normal in certain species (notably the sheep and pig). Bremer was impressed by the fact that of the 80 human embryos in the Harvard Embryological Collection ranging from 5 to 45 mm, four showed a tracheal bronchus. He feels that many of the tracheal buds disappear by resorption during development and that this may account for the presence of certain tracheal cartilaginous irregularities or defects seen so commonly in postembryonic life.

AGENESIS OR HYPOGENESIS OF THE LUNG

Schneider[7] has classfied pulmonary agenesis into three categories, depending upon the stage of development:

1. True aplasia (no trace of lung, bronchus, or vascular supply) (Figs. 20, 21 and 22).

2. Tiny outpocketing (bud) of the trachea, with no lung tissue (Figs. 23 and 24).

3. Extreme hypoplasia of the bronchus with no true lung tissue (Fig. 25).

In most instances the pulmonary condition is relatively asymtomatic, and the abnormality is found only on examination. In 1944 five cases of agenesis of the lung were reported from The Children's Hospital, Boston, all diagnosed during life, and four of these patients are still living normal lives.[5] Each of them showed, in addition to the pulmonary condition, other congenital defects though most of these were of a minor nature. The one patient who died was a four-months'-old infant and this death from pneumonia occurred before

Fig. 25.—Agenesis of left lung (age 6 months). AP view. Note hypogenesis of left bronchial tree, with no lung tissue.

the antibiotic era. This patient had a patent ductus arteriosus and hypoplasia of the liver, spleen, and kidneys.

SUMMARY

Our experience with the diagnosis of congenital anomalies of the tracheobronchial tree, and of the aortic arch compressing the trachea and esophagus, leads us to the conclusion that these anomalies are far more common than was formerly supposed. Inspiratory stridor due to alteration in caliber of the tracheal airway, and not related to any laryngeal abnormality, was a common finding in patients with tracheal compression. This confirmed our belief that

examination of the trachea, as well as of the larynx, should be carried out in patients with stridor where the slightest suspicion exists that the larynx is not the source of the trouble. We are convinced that the use of general anesthesia, properly administered, has allowed us to perform more searching and complete endoscopic and Lipiodol® studies on our patients, with a minimum of trauma and a maximum of safety, than would have been possible otherwise. Though most of our patients were in infancy or early childhood, subglotic edema of clinical significance was rarely noted, and in no instance was tracheotomy required. Lipiodol, as a contrast medium may, in our opinion, be used in regulated amounts, in even the smallest infants, when procedures are carried out quickly and the oil is removed by suction immediately after x-ray studies are made. Sensitivity to lipiodol must be very rare indeed since we have never met with any recognizable symptoms of this in the many thousands of contrast medium studies done here during the past eighteen years.

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DISCUSSIONS

Dr. Lyman Richards (Wellesley, Mass): In my early high school days we suffered under an irascible Latin teacher who would tolerate only one response from a pupil who had not done his homework, and that was "not prepared." When I came here this morning, I was very much in that position, because I didn't even know I was going to discuss this paper, much less have any opportunity to prepare myself. Fortunately, Dr. Ferguson tipped me off this morning, and that gave me an inkling of what was ahead of me.

It is also fortunate that it is a paper which obviously doesn't admit of any very critical discussion. It is hard for me to realize that it is almost 25 years ago

since Dr. Ferguson and I—he then playing the role of the very promising house officer, used to put lipiodol down the lung in more or less hit-or-miss fashion. We thought we had done pretty well to differentiate between lung abscess and bronchiectasis. I am sure that many of these interesting anomalies were missed in those days, and we failed to come up with an explanation for various clinical signs of pulmonary pathology and obstruction which he is now bringing to light.

Also, we lack the skill of a Dr. Gross—incidentally, also a house officer of mine, though I'm sure his present success has no connection with that event.

But thoracic and pediatric surgery have obviously made amazing strides since that time, and with this interesting help which Dr. Ferguson and Dr. Flake can now give the pediatric surgeon, I am sure that much is being accomplished which we failed to achieve in former days.

It would be perfectly futile and unwarranted of me to consume your time by making any critical comments about these pictures.

One thing Dr. Ferguson failed to tell you (with his innate modesty) was that this demonstration in Mexico City won a gold medal, and I'm sure that those of you who haven't seen these pictures before will feel that was well deserved. It is a classic example of how someone starts with a procedure in a somewhat primitive way, trying to do the best he can, and then, over a period of years, with a chance to look back over that time, as is true of so may other fields of medicine—progress goes on, and something is done better and better all the time, and still there is always a goal ahead to achieve. With my knowledge of how some of this work started at the Children's Hospital, I am sure that Dr. Ferguson and Dr. Flake have made a very striking contribution to the study of pulmonary disease in children.

DR. FRANCIS W. DAVISON (Danville, Pa.): This is beautiful work and I have only one question. I would like to ask Dr. Ferguson whether or not he worries about the fire and explosion hazards? That question comes up, and I'd like to have him tell us about his precautions, if any.

DR. CHARLES F. FERGUSON (Boston, Mass.): I should like to thank Dr. Richards for his very kind words, and Dr. Davison for his comment.

We usually stop the anesthesia and put a wet towel over the patient's face when he is in the fluoroscopic room, and he is down just enough to maintain satisfactory anesthesia for the plates. We have a very "rapid-fire" technician who takes these plates, and he can do five or six films in two minutes, or something like that, slipping the cassettes under the patient very quickly. We don't have the actual ether in the room at the time.

LXXIX

POSTOPERATIVE ESOPHAGEAL STENOSIS

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AND

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There has been great progress in the field of esophageal surgery during the past 15 years. The advances that have taken place are due to several factors. There is a better understanding of the anatomy and physiology of the esophagus. Chemotherapy and antibiotic therapy have helped greatly in the reduction of operative mortality. The ingenuity and imagination of the thoracic surgeon have brought about improved technical skill and perfection of procedures that were once thought to be impossible. Almost any disease of the esophagus can now be approached by the surgeon with little fear of technical difficulties or operative mortality.

It is to be expected that when new surgical procedures are first employed, some patients will not have a satisfactory result. The surgical procedure may be successful but the patient continues to have symptoms. The latter are sometimes worse than the original complaint.

There is a never-ending succession of patients with esophageal disorders. With the increased span of life, more and more patients live to an age at which esophageal diseases are not uncommon. When esophageal surgery was begun in earnest, there was a vast amount of clinical material. Many types of operations were devised and attempted. Not all were successful. Much knowledge was obtained by a study of the end results of such surgical procedures. New techniques and new approaches were constantly being developed. However, it gradually became an established fact that surgical correction of diseases of the esophagus was not always satisfactory. The lack of success was not due to poor technique, infection, or to oper-

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ative mortality. In most instances, a poor result was due to failure to get rid of the original disease such as carcinoma, or a persistence of the old or development of new symptoms after a benign condition had been surgically corrected.

The lower third of the esophagus is a frequent site of disorders. The latter include hiatal hernia, cardiospasm, ulcer of esophagus with peptic esophagitis, epiphrenic diverticulum and carcinoma. Differential diagnosis is determined by a careful history, physical examination and x-ray study of the esophagus, including careful fluoroscopy with positioning of the patient and esophagoscopy. Putney and Clerf,[1] have stressed the importance of insufflation of air into the lower esophagus as an aid in diagnosis.

All of the above have been subjected to a variety of surgical procedures. The data on the results of surgery in this region point strongly to need for maintaining a physiologically active cardia. Any surgical procedure which disturbs the normal valve mechanism of the esophagogastric junction may produce severe complications. The latter include reflux with resultant esophagitis, ulceration, hemorrhage and, occasionally, stenosis of the lower esophagus. An important factor is the susceptibility of the esophageal mucosa to the erosive action of acid pepsin and, to a lesser degree, the intestinal juices.

Postoperative stenosis of the esophagus has been seen in 15 patients who had surgery for cardiospasm, varicose veins of the stomach and lower esophagus, carcinoma of the stomach, carcinoma of the esophagus, lye stricture and congenital tracheo-esophageal fistula (Table 1).

All of these patients were examined by esophagoscopy. The findings varied depending in part on the original lesion. In patients with carcinoma, the region of the anastomosis was the site of local recurrence or residual tumor. This was indicated by ulceration with a positive biopsy or compression stenosis.

Shelving of the lumen below the line of anastomosis was present in two cases. The passage through the stenosed area took a sharp turn almost at a right angle. This made any method of dilatation very difficult to perform. In one instance, the shelving was so great that a bougie which was passed into the stenosed area disappeared from sight and broke off. It was recovered three weeks later in the esophagus. Apparently it had stuck in the tortuous part of the stenosis and was regurgitated into the esophagus.

Varying degrees of esophagitis and ulceration were observed through the esophagoscope. Congestion, redness and increased fra-

TABLE 1.

POST-OPERATIVE ESOPHAGEAL STENOSIS.

DISEASE	OPERATION
1. Hiatal hernia	Esophagogastrostomy
2. Hiatal hernia	Esophagogastrostomy
3. Hiatal hernia	Esophagogastrostomy
4. Carcinoma of esophagus	Esophagogastrostomy
5. Carcinoma of esophagus	Esophagogastrostomy
6. Carcinoma of esophagus	Esophagogastrostomy
7. Carcinoma of esophagus	Esophagogastrostomy
8. Carcinoma of esophagus	Esophagogastrostomy
9. Carcinoma of esophagus	Esophagogastrostomy
10. Varices of stomach and lower esophagus	Gastrectomy and Esophagojejunostomy
11. Varices of stomach and lower esophagus	Gastrectomy and Esophagojejunostomy
12. Congenital tracheoesophageal fistula	Esophagoesophagostomy
13. Congenital tracheoesophageal fistula	Esophagoesophagostomy
14. Lye stricture lower ⅔ of esophagus	Esophagojejunostomy
15. Cardiospasm	Esophagoplasty, Finney type, transabdominal vagotomy and gastroenterostomy

gility of the mucosa were fairly common findings. The passage of the tip of the tube would occasionally peel off the mucous membrane. Bouginage sometimes caused brisk bleeding.

It is of significance that the esophagoscopic appearances of the stenosis did not change with the passage of time. As a rule the same findings were present as often as the patient was examined even several years after the original operation.

The surgical correction for two cases of congenital tracheoesophageal fistulae consisted of an esophagoesophagostomy. The cardioesophageal junction was not disturbed. The strictures which developed responded readily to dilatation. In one patient it was necessary to do retrograde bouginage.

There were 12 cases in which the operative procedure included removal of the cardio-esophageal junction. These patients had either an esophagogastrostomy or an esophagojejunostomy. The stenoses which were present developed at the line of anastomoses or close to it. Each of the patients had esophagitis and reflux of gastric or intestinal juices into the esophagus. Hemorrhage and ulceration were common findings. The patients all had symptoms of varying degrees including, heart-burn, pain and malnutrition as well as dysphagia.

The remaining case of stenosis was in a patient who had cardiospasm. A Finney type plastic procedure was performed without benefit. This was followed by a vagotomy. Anatomically, her widely dilated esophagus became narrow, but at the cardioesophageal junction, she developed a severe esophagitis with ulceration and stenosis. She had severe hemorrhages and on one occasion, had to have a blood transfusion.

Postoperative esophageal stenosis is difficult to treat. The patient not only complains of dysphagia, he also has many other symptoms. The latter may be more distressing than his original disease. In addition to dilatation of the strictures, it is also necessary to give constant medical care to the patients. They all have nutritional disturbances and therapy for the most part is symptomatic. Each patient is an individual problem.

The patient who has a benign postoperative esophageal stricture as a result of esophagogastrostomy, or esophagojejunostomy is apt to be a patient as long as he lives. The group presented is not large, but when one considers that the same patient is due to return at either weekly or monthly intervals for dilatation, the problem is a formidable one. This is particularly so in view of the fact that such patients are on the increase due to the greater number operated upon.

An editorial in *Surgery, Gynecology and Obstetrics*[2] dicusses the damaging result of esophageal reflux following surgery for benign conditions. It is pointed out that the patients are frequently worse off than those with malignant conditions because they live longer. The importance of the esophagogastric valve mechanism in surgery of the esophagus and stomach is stressed.

Allison[3, 4] has made a thorough study of the lower esophagus with special attention to the repair of hiatal hernia. He feels that the sliding hiatal hernia is amenable to repair which should bring about the restoration of a physiologically active cardia. The rationale of surgical treatment should be to cure the deformity which allows

acid to reach the esophagus. He sutures the esophagogastric ligament to the undersurface of the diaphragm, and repairs the hiatal defect posteriorly in an attempt to restore normal anatomic relationships. His results have been good. His patients have fewer postoperative complications.

Stensrud[5] discusses the factors which prevent regurgitation into the esophagus. His results on repair of hiatal hernia have been better with the technique of Allison.

Kay[6] describes the inferior constrictor of the esophagus as being a thickening of the inner muscle layer just above the hiatus. He cites the conditions under which the constrictor becomes hypertrophied. He has demonstrated it in patients with achalasia and recommends a myotomy. Kay further describes the opening and closing mechanism of the lower esophagus as interrelated between the diaphragmatic esophageal ligament and the inferior esophageal constrictor.

The papers of Allison and Kay emphasize the importance of maintaining the esophagogastric junction when surgery of the lower esophagus is performed. The prevention of postoperative esophageal complications lies in adherence to their principles.

Surgery for benign diseases of the esophagus should be restricted to patients whose symptoms will not respond to conservative therapy. The surgical procedure selected should be one that will allow the esophagus to function without the development of esophagitis and postoperative stenosis.

A benign stricture should never be operated upon if it can be treated by esophagoscopic dilatation or by bouginage. Finnerty[7] presents the problem of surgery for caustic strictures of the esophagus. He presents six possible operations that might be performed.

It is of interest that at a recent meeting of the American Association for Thoracic Surgery five papers on surgery of the esophagus were presented. Holinger, Johnston, Potts and da Cunha[8] reported on more than sixty patients, adults and children, with postsurgical strictures of the esophagus. Watkins, Prevedel and Harper[9] reported a method of preventing peptic esophagitis following esophagastrostomy. They make a flutter valve mechanism in the esophageal portion of the esophagogastric junction.

SUMMARY

Postoperative esophageal stenosis is a frequent complication of esophageal surgery.

The most common complication is esophagitis with ulceration and hemorrhage at the site of the anastomoses. Reflux is probably the cause of the esophagitis.

Surgical correction of benign lesions of the esophagus should be performed only when conservative measures fail to alleviate the condition.

In surgical procedures on the lower esophagus the preservation of the cardioesophageal junction with restoration of an active physiological valve mechanism should be considered.

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DISCUSSIONS

Dr. Louis H. Clerf (Philadelphia, Pa.): Mr. President, members of the Association and guests: One of the problems presented by Dr. Baker and his associates is met with in cases of hiatal hernia. They are afflicted with failure of the normal pinchcock mechanisms to protect the esophagus.

The thoracic surgeon, in his endeavor to correct carcinoma of the esophagus, achalasia and other conditions, has produced the same condition from which the patient with hiatal hernia suffers.

I well recall Dr. Chevalier Jackson discussing the value of the pinchcock at the lower end of the esophagus and its importance in preventing gastric juice from getting into the esophagus and producing peptic ulceration. We have met up with a number of these cases, particularly since thoracic surgeons have become more energetic and we have referred to them patients we believed could be benefited by their ministrations.

It is admittedly true that they have done a fine job in correcting things mechanically, but often these patients present themselves to us later with new difficulties, and these have indeed been problems.

Probably our greatest problems occur in the cases of achalasia, which, following partial esophagogastrectomy exhibit not only difficulty in swallowing but pain and hemorrhage. Neither of these conditions can be corrected esophagoscopically, since in many there is no stenosis.

The problem of reflux esophagitis is an exceedingly difficult one to correct, and so far as I am concerned, in the absence of stenosis, there is little we can do save admonish the thoracic surgeon that he should, if possible, maintain or leave undisturbed the integrity of the hiatal pinchcock. I am not sympathetic to referring a patient with achalasia to a thoracic surgeon if he is contemplating doing a resection which includes the hiatus because we have practically no patients in that group who are symptomatically comfortable.

In many instances, by using the old Plummer dilator passed over a previously swallowed thread, we are able to render patients reasonably comfortable without hemorrhage and without the symptoms that we describe as those of reflux esophagitis. The men who are now doing our cases pursue a surgical procedure which preserves the hiatal sphincter.

Carcinoma is an entirely different problem, because there the surgeon wishes to save the patient's life and he is justified in anything he does to accomplish this. It is up to us to help him as much as we can to main the integrity of the food passage ways. We now have under treatment a number of operated cases of carcinoma whom we dilate just as we have been dilating cases of cicatricial stenosis over the years.

With regard to cicatricial stenosis, I am disturbed by the thoracic surgeon who considers that the best plan of therapy is to resect the cicatrized esophagus and bring the stomach up into the chest cavity. Mechanically, it is fine; functionally, I am not in agreement because some present problems. They suffer from reflex esophagitis as well as other disturbances.

My one thought in connection with this whole business is that whereas we are perfectly willing to help the thoracic surgeon, we would beg him to bear in mind that when he is doing some of these procedures that have for their purpose removal of part of the stomach and esophagus, he is really handing us back a case of hiatal hernia which is going to develop a lot of symptoms, and for which we can do amazingly little. We would like to have him contemplate perfecting his technic so he can supply aids not requiring the removal of the hiatus. We ought to preserve the integrity of the hiatus whenever we possibly can.

Dr. Paul H. Holinger (Chicago, Illinois): Mr. Chairman, members of the American Broncho-Esophagological Association and guests: I do appreciate Dr. Baker's bringing this subject to our attention. His presentation was a most excellent and complete one of a subject which I think is increasing in frequency, and is probably one of the most difficult problems that we have in esophagology at the present time, both in number of cases and the rapidity with which they are increasing.

I would like to summarize our findings, if I may, for the purposes of the record.

At the recent meeting of the American Association for Thoracic Surgeons we presented 101 cases of esophageal strictures following surgery of the esophagus. There were 26 cases of congenital atresia with tracheo-esophageal fistula, which had extreme stenosis of the esophagus requiring dilatation. Half had been referred after surgery elsewhere, and the remainder represent about a quarter of the living cases of esophageal atresia and tracheoesophageal fistula that we have at Children's Memorial Hospital.

The second group of these 101 cases consist of stenosis following resection of benign stenosis; there were 23 cases of such stenosis—5 with stenosis following resection of a congenital stenosis, 3 with stenosis following resection of lye strictures, and 15 following resection of chronic inflammatory stenosis, such as peptic ulcer of the esophagus, 8 cases, and various procedures for cardiospasm, 7 cases.

There were 9 cases of stenosis following surgery for non-stenosing lesions of the esophagus. That almost seems ridiculous, but it actually occurred; in 7, stenosis followed a diverticulectomy (in one a complete closure of the esophagus had been done, requiring gastrostomy, and eventually working up from below and down from above to find a lumen through the area. In taking off the diverticulum, the surgeon had apparently sewn the esophagus closed rather than sewing only the opening created as the diverticulum was removed). There were two with stenosis of the esophagus following thyroidectomy; the thyroid had extended substernally, and there had been a resection of a portion of the esophagus to cause this stricture formation.

The fourth group is that of resection for carcinoma. There were 43 cases in this group, with extreme stenosis of the esophagus; of course, as Dr. Clerf and Dr. Baker mentioned—this problem is secondary to the importance of the carcinoma itself, and yet the analysis of these cases demonstrates that for the most part they occurred in cases of carcinoma of the upper or lower thoracic esophagus in which an esophagogastrostomy was performed, with resulting lack of the sphincter action. There were 15 carcinomas of the cervical esophagus, where resection resulted in marked stenosis requiring post-operative dilatation.

In analyzing these cases, I think we can very much agree with Dr. Baker's remarks. Resection of an hour-glass stricture of the esophagus, which is so simple a technical thoracic surgical procedure and appears so obvious a mechanical solution, is certainly not the final answer to the problem. It is too often followed by reformation of the stricture, requiring the same procedures that the patient had beforehand. Infection, of course, increases the probability of stricture formation. Certainly, technic is a factor—the use of very fine silk and very careful anastomosis.

Thus, if part of the esophagus is removed, and an esophagogastric anastomosis is made, strictures form; in those cases in which the more extensive procedure of jejunal esophageal anastomosis is used with the Roux-y type of procedure, or a procedure in which the gastric-secreting portion of the stomach is reduced, the patients have by far the better prognosis. For these reasons, after analyzing such cases, I think we must go back to our thoracic surgeon friend and insist that the simple procedure of removal of a segment of the esophagus and direct reanastomosis is obsolete unless associated with some means of either removing the gastric-secreting portion of the stomach or maintaining the function of the sphincter itself.

Dr. Edward B. Benedict (Boston, Mass.): Mr. President, members and guests: Our experience at the Massachusetts General Hospital has been definitely more optimistic than that so far reported on the subject of postoperative stenosis of the esophagus.

To my mind it is convenient to classify postoperative esophageal stenosis into four categories, depending upon what type of operation was done preceding the stenosis.

1. In the first category are cases that followed major surgery of other types, not on the esophagus. *Esophageal stenosis may follow any major surgical procedure,* especially if there has been vomiting or regurgitation of acid gastric secretion. In a series of a hundred cases of esophageal stenosis published by Gillespie and me in the April 1954 number of S. G. & O.,[1] there were 22 patients who had

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had a major surgical procedure within five months prior to the development of dysphagia. Of these 22 patients there were 6 gastrics, 5 gallbladders, 4 involving the appendix, colon or rectum, 2 lobectomies and 5 miscellaneous. An inlying gastric tube had been used in 5 cases. Most of these patients have responded well to bouginage.

2. In the second category, *after total gastrectomy for carcinoma*, there have been a few benign esophagojejunal stenoses, and a few with recurrent cancer of the stoma, responding well in the benign cases to bouginage. We examine them first by esophagoscopy, with bouginage at that time; later we have them swallow a thread and carry out further bouginage by mouth using the thread as a guide. I think the transthoracic route is very much better for total gastrectomy than the abdominal route, because of better exposure, better margin and better mucosal approximation. We have had no postoperative stenosis in the group approached transthoracically.

3. In the third category, *after resection for cancer of the esophagus*, I know of only one case of postoperative benign stenosis. This occurred 11 years ago, and was probably due to poor technic in mucosal approximation. This patient responded to bouginage over a one-year period, and has been symptom-free for the past ten years. There have been a few recurrent cancers of the esophagus near the esophagogastric junction, where the surgeon was unable to get high enough above the original cancer.

4. In the fourth category, *after esophageal resection for benign peptic stenosis*, in the 100 cases which we reported, there were 20 surgically resected; the other 80 were all treated medically and by bouginage. Of the 20 resections, 2 had recurrent stenosis, 1 was relieved by a second operation, and the other was relieved by medical treatment and bouginage.

Dr. Richard Sweet has done most of the esophageal surgery in these cases. He and I believe that recurrent stenosis can usually be avoided by resecting well above the area of esophagitis and getting very careful mucosal approximation with very fine silk sutures.

With regard to achalasia, it has been pretty well shown that the Heller operation is the best. That is essentially the same as a Ramstedt operation, only it is performed at the cardia, merely splitting the muscle. It has been quite successful. I agree that resections of the cardia for achalasia have resulted in immediate relief of the dysphagia, but other symptoms have developed, especially esophagitis and gastritis with hemorrhage.

Dr. Gabriel Tucker (Philadelphia, Pa.): Gentlemen, to me this has been a most interesting discussion. So much time has been taken up that we don't have enough time to go into details, but I would like to say that for cardiospasm the use of the Heller operation has proved very satisfactory, and there have been no complications. You still have a sphincter at the lower end of the esophagus.

We have had about 25 cases at the Graduate Hospital of the University of Pennsylvania in the past year. The only patient having any regurgitation was one who had been operated on some 20 years ago, when all the nerves of the lower end of the esophagus had been resected, with the idea that this would affect the action of the lower sphincter. That patient did have some reflux, but the other patients have done well.

You all know what the Heller procedure is. It is the same thing that is done in pyloroplasty. You cut down through the muscle and let the mucous membrane bulge. That is practically all that is done. You don't sew it up again. It does relieve these patients, and gives excellent results in cardiospasm.

We have followed these patients for a number of years, and they have done well.

There is one other thing I would like to suggest about cases in which anastomosis has been done between the esophagus and the jejunum, particularly when the entire stomach is removed. We have seen the formation of stenosis, but we have a very simple method of keeping it open that has been effective. We put in an indwelling bougie when we no longer get the results that we ordinarily get by the rapid dilatation through the esophagoscope, and we leave it in over night or even longer. It is suspended by a string through the mouth, and during the time the hollow bougie is in the esophagus, the saliva and anything taken by mouth goes into the stomach. This puts the area at rest, and, of course, with the jejunum, you don't have the regurgitation of acid as you would with the entire stomach removed.

This keeps the passage open until you get to the point where you let the patient swallow a mercury bougie. My friend, Dr. Richards, has had a lot of experience with mercury bougies and he, like Dr. Clerf, wants to pass these patients on to his successor. It has given us a great deal of satisfaction to keep this open.

I feel that one mistake sometimes made is that we don't put in an indwelling bougie soon enough; we wait until the esophagus is closed off almost completely, and then you can't use the indwelling bougie.

I have a little film on the esophagus, and Dr. Broyles says that I might show it tomorrow. It is in connection with the mechanism of swallowing. At that time I will demonstrate several of these cases for which we have used an indwelling bougie followed by the mercury bougie, and these patients have gotten along very nicely.

Of course, patients having a resection of the stomach for cancer may have late bony metastases. We had such a patient who was swallowing normally after the method we have described, but he developed metastases in his bones. Well, that is not a matter for the esophagoscopist; that is something we can't limit.

I have talked about this so much that I'm almost ashamed to mention it, but tomorrow we would like to show you the film, to show you the way the method works.

I want to congratulate the other speakers. They all did a very good job, especially Dr. Baker and Dr. Clerf. This is a real problem, and we appreciate the difficulties.

DR. DANIEL C. BAKER, JR. (New York, N. Y.): I thank Drs. Clerf, Benedict, Holinger and Tucker for their comments.

I have nothing to add to what Dr. Clerf and Dr. Holinger had to say.

With regard to Dr. Benedict's remarks about carcinoma of the esophagus, I have had no experience of a benign stricture of the esophagus following surgery for carcinoma. Each patient that I have seen has had a recurrence. We have no patients alive at the end of five years.

I have had no experience with Dr. Tucker's method of using a hollow bougie for the prevention of stricture. I certainly think it is worthy of a trial.

LXXX

CARDIAC ARREST FROM THE POINT OF VIEW OF PREVENTION

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References to cardiac arrest are appearing with increasing frequency in medical literature. The emergency treatment recommended, immediate, intrathoracic, cardiac massage, cannot be taken lightly. It would appear important, therefore, to stress etiological causes, and to avoid, if possible, this catastrophic eventuality.

A review of the literature respecting cardiac arrest, or what is more accurately termed, "sudden standstill with gray cyanosis" suggests that the precise cause or causes are unknown. However, certain predisposing factors are well recognized:

1) Pre-existing cardiac disease.

2) Hypoxia from stagnant, anaemic, anoxic and histotoxic anoxia, singly or combined.

These predisposing factors can be triggered by: cyclopropane, chloroform or ethyl chloride, epinephrine, injected or autogenous, barbiturates plus cyclopropane, vagus stimulation, cardiac and pulmonary surgical trauma, drug sensitivity (cocaine), electrolytic imbalance.

There is a general feeling that a combination of the above factors, existing simultaneously, may result in cardiac arrest. Endotracheal intubation has been referred to as a causative factor.[10] As this technique is mandatory in head and neck surgery, and since endotracheal intubation is a necessary preliminary to bronchoscopy, it occurred to us that it might be of value to inquire into this possible etiological factor. The following questions were, therefore, placed before the membership of the American Broncho-Esophagological Association: 1) Have you encountered cardiac arrest in the course of routine bronchoscopy? 2) Approximately how many bronchoscopies does this experience represent?

Forty-four inquiries resulted in eighteen replies. These replies represented more than one hundred thousand bronchoscopies. Not a single case of cardiac arrest was reported in routine bronchoscopies.

The complexity of the problem, involving as it does the integrated activity of the cardiovascular, respiratory and nervous systems, not infrequency reflects in the words of Dr. Arthur Guedel,[7] "the misapplication of misinterpreted pathological findings arrived at by a faulty technique." And to quote Dr. Raymond Schulz: "Laboratory theories should not be rushed into the clinic too quickly. When trouble occurs, you can be sure that there has been an insult somewhere. Is it nerve ganglia, mechanical, or toxic? Ether still seems to be the safest. Its slow induction gives the vasomotor control a chance to adjust itself. Are not some of the speed demonstrations of modern anaesthesia comparable to the sudden and instantaneous introduction of a urethral sound into a man's bladder by an expert, producing death by shock? This was done as a display of skill, but the patient's mechanism could not take it. Then also, the complex premedications may be disturbing. A portion of the nervous system is anaesthetized and the autonomic reflexes are not. A sudden physical insult can still have its effect on the nerve centers not anaesthetized. . . . Cyclopropane is a cyclic compound, bound between carbon atoms possibly strained c3H6. We know that benzine is toxic C6H6, the double bonds therein possibly less strain in the chain. Much is still not understood."

INCIDENCE OF CARDIAC STANDSTILL

The incidence of cardiac stand-still has been variously estimated as: "once in a surgeon's lifetime,"[1] "seventy cases collected,"[2] "one in twelve hundred in poor anaesthetic risks."[3] Everyone will agree that an accident occurring to one near and dear to us is always statistically 100 per cent. There is, furthermore, no disagreement in the principle that every known measure should be taken to avert and to treat such a calamity. Nevertheless, the incidence of the mortality noted above, and the incidence of asphyxial death (at least 50,000 a year) should be recognized. Emphasis should be placed upon the accident with the greater mortality.

Fortunately, as has been noted above, hypoxia is already recognized as a basic etiological factor in cardiac arrest.[5, 11] To prevent and to correct hypoxia is, therefore, a direct means of reducing the incidence of cardiac stand-still. In a recent flight to Europe, the writer visited the deans of the medical schools in Dublin, London,

Paris, Heidelberg, Rome, Barcelona, Madrid an Lisbon. The purpose of the visit was to stress the importance ot clinical hypoxia. It was pointed out that induced unconsciousness, with reflex and muscle control, is the logical approach to the three-dimensional study of cellular, cardio-respiratory and cerebro-spinal hypoxia. General anaesthesia in the human being is an essential discipline for all physicians.

The resident and, presently, the general practitioner who has failed to become acquainted with general anaesthesia, has suffered an irreparable loss. He is no longer familiar with the unconscious patient, and with the management of the pathologic physiology which accompanies, or which may precipitate unconsciousness. He is no longer sensitive to "man . . . alive," the three-dimensional view of form, function and time. This sensitiveness comes only when one is required to control this integration in the unconscious man—the man in whom induced control has taken the place of voluntary or automatic control. If one is not responsible for, and obliged to direct the factor of timing, this third dimension is likely to be overlooked altogether. Time operates by determining the instant, the degree and the duration of function. Its recognition allows the instantaneous freezing of the fluidity of function. It permits a precise diagnosis, or the application of a desirable act, as it were, in functional transit. The effects of a gas or a drug either progresses or regresses. It is never static. Timing determines the correct instant of effect or action. Form and function may be taught. Time must be experienced.

An attempt will now be made to illustrate what is meant by the three-dimensional point of view as applied to the physiopathology of clinical hypoxia. This approach answers the three questions: What? How? When? Let us consider briefly the anatomy, the form or the "What?" of our trilogy.

For practical purposes the heart and the arterial system may be considered as a unit. In this conception the heart emerges as a twisted loop of the major artery. The muscle coat of the artery, greatly increased in bulk, provides power to propel the blood stream. Intercepting one-way valves, in the chambers of the heart, provide and maintain direction to the impelled stream. The cardiovascular system terminates in the lungs and in the tissues. In the lungs, the capillary system, through more than 750 million alveolae, presents an area 100 times that of the surface of the body. In the tissues, the capillaries permeate all organs, muscles and structures. In the two major areas noted, return flow takes place through venules and veins

of ever-increasing caliber. These vessels complete the circle by discharging their contents into the right heart.

The respiratory system, enclosed in a bony cage capable of expansion and contraction, is limited by the muscles of the neck and upper airway, anteriorly by the sternum and the clavicles, posteriorly by the vertebrae; laterally by the ribs, and the intercostal muscles and below by the diagram. Nerve centers, ganglia, trunks and nerve endings transmit the functional energy in the lungs through the vagus and the alveolar nerve endings in the cardio-vascular system through the aortic arch and the carotid sinus, sympathetic and parasympathetic ganglia and nerves, and through the deep and superficial cardiac plexus, in the heart, through the sinu-auricular node, the bundle of His, and the heart muscle nerve endings. Blood chemistry reveals varying concentrations of CO2, lactic acid, sympathin and parasympathin.

Function, or the "how?" These cardiovascular-respiratory-nerve structures operate as an interdependent, synchronized unit. One portion cannot be isolated from another. Difficulties which develop in one portion are instantly reflected and offset by compensating responses in the others. There is, of course, a physiological limit to these physiological compensations which can be, and often are, broken in the unconscious patient by drugs, mechanical factors and trauma. Increased demands on function are more readily met than reduced or complete cessation of function.

Cardiac rate and rhythm are regulated by afferent and efferent impulses, and by the chemistry of the blood. Impulses from the sympathetic and parasympathetic ganglia flow by nerve trunks to accelerate or retard rhythm. Superficial and deep cardiac plexuses are all parasympathetic (slow to the point of arrest). CO2 in alveolar air, CO2 and lactic acid in the blood, acting on the carotid sinus and aortic arch, are reflected in the dilatation and contraction of the arteriovascular bed.

The above references to form and function are common knowledge. They are noted here in order to stress their detachment from reality. They present merely facts. The living organism is not recognizable in a mere recitation of these facts. The third dimension, timing or "When?" is essential. Timing presents the organism as alive, acting and reacting to acceptable or to destructive stress. For the purpose of demonstrating this third dimension, a sound motion picture of the living dog's heart, and its reaction to progressive hypoxia has been prepared for this meeting.

A dog was anaesthetized by intravenous nembutal. He was intubated. A double thorocoplasty and pericardostomy were performed. A canula was placed in the carotid artery. The blood was prevented from coming into contact with the mercury in the manometer by a buffer of saline solution. The esophagus will be seen to run across the top of the field. The retracted edge of the pericardium is seen above the auricles. Respiration is carrid on by a mechanical pressure pump. The lungs expand rhythmically in the ambient atmosphere. The stop watch in the field will be frequently referred to to mark a precise point in the events to take place.

The lungs are filled with oxygen. Artificial respiration is discontinued. It will be recalled that a man of 70 kg has a cardiac output of approximately 5 liters. A dog of 10 kg has a cardiac output of approximately 2.5 liters. A man's cardiac output is, therefore, only about 1/3 that of a dog. The stop watch now indicates 3 minutes, 40 seconds. Note the arrythmia and the physiological dilatation of the heart which has appeared.

Artificial respiration has been resumed. The clinical material which you observe is similar to that which Harvey first utilized three hundred years ago, and which resulted in his discovery of the circulation of the blood. Harvey used a foot bellows to maintain artificial respiration. In his initial experiment, however, he found the rate of the heart beat of the dog so rapid that he was obliged to turn to the slowly beating hearts of the cold-blooded animals. We are now about to enter the terminal phase of hypoxia. This period of our experiment required 17 minutes, 40 seconds. It is completed in the film in 3 minutes. The beginning of the terminal hypoxia is marked by the stop watch at zero.

Artificial respiration has now been permanently discontinued. At the end of 3 minutes, the ventricular beat will be slowed. Contractions will be violent. Accumulating cyanosis will be observed. While our attention is focused upon the heart as an isolated organ, it must be born in mind that the unseen circulatory, respiratory and neurological factors involved are of equal importance.

In the early stages of acute progressive hypoxia, the immediate effects are those of blood pressure and changes in the chemistry of the blood acting on the aortic and carotid sinuses. Later the effects are on the heart muscle. Progressive hypoxia is clocked at intervals. The last ventricular beat occurs in 7 minutes 35 seconds. The last auricular beat in 17 minutes 40 seconds.

Complete heart block has now developed. The coronary circulation is greatly engorged. The auricles are distended. Pathological dilatation has taken place. Irreversible cerebral damage has occurred. Blood pressure has dropped to zero.

We have observed these phenomena through the integrated factors of form, function and timing. The cessation leaves us with cellular and somatic death.

The experiment which you have seen has demonstrated the ability to freeze the fluidity of function, and to pinpoint a precise observation, i.e., the last ventricular beat, the development of heart block, the last auricular beat. It presents an appeal which does not exist in the observation of form and function alone. Drama lies in the satisfaction and in the force which the combination of form, function and timing afford in the study of a living organism, our patient. In your surgical work, as in this demonstration form and function may be taught. Timing must be experienced.

Clinical hypoxia cuts across the entire field of medicine. It cannot be departmentalized. The problem is not an anaesthetic

problem, because the anaesthetist is familiar with the technique of treatment. It is a bronchoscopic problem, an obstetrical problem, a pediatric problem. Your skilled knowledge and your broad viewpoint is sought. It is needed to help your medical school and your hospital medical board to organize means to prevent and to treat clinical hypoxia. The speaker is expendable in the promotion of this project. Your support is important.

CONCLUSION

No clinical evidence has been advanced to suggest that bronchoscopy (endotracheal intubation) per se precipitates cardiac arrest.

The literature respecting arrest of the heart is practically unanimous in declaring that hypoxia, per se, produces sensitization of the heart muscle which may be triggered to fibrillation or arrest by a variety of causes, e.g. epinephrine, injected or autogenous, or by direct, pulmonary, cardiac or other deep reflexes occurring in operating procedures.

The first and immediate treatment is endotracheal insufflation of oxygen under sufficient pressure to excite the Hering-Bruer reflex.

Pre-operative and operative medication which contributes to stagnant, anoxic or histotoxic anoxia should be avoided.

Form is dead, function is mechanical but timing is life in action, struggling to meet the demands of pathological stress.

If we can but take away with us an understanding of the enormous handicap which we place on the integrated activity of the living organism by blocking compensatory mechanisms through the multiplicity of depressant drugs, by the shock of the unanesthetized autonomic reflexes and by trauma, the calamity of cardiac arrest is not so likely to occur. The present high mortality from hypoxia will be reduced.

2 EAST 63 STREET.

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LXXXI

PULMONARY ALVEOLAR ADENOMATOSIS

REPORT OF FIVE CASES

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AND

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In recent years more and more attention is being focused upon a relatively uncommon disease in man, variously called pulmonary adenomatosis, or alveolar cell carcinoma, which bears considerable resemblance to an epizootic disease of sheep frequently referred to as jaagsiekte, pulmonary adenomatosis, epizootic adenomatosis and Montana progressive pneumonia of sheep.

This disease in sheep is characterized by extensive inflammatory changes in the lung, associated with proliferative changes of the lining cells of the alveoli. These lining cells, which are epithelial in appearance, vary from cuboidal to columnar in arrangement and are not ciliated and do not appear to be continuous with the bronchial epithelium. In the advanced stage there is considerable papillary infolding into the alveoli, producing the histological configuration of an adenoma or adeno-carcinoma, from which, however, it differs in that it maintains a regular cellular pattern and is not believed, by many investigators, to metastasize. However, Aynaud, quoted by Dungal,[1] described a case of jaagsiekte in sheep in which metastasis occurred.

Neuberger[2] and others have also demonstrated evidence of metastasis in human cases included as examples of pulmonory adenomatosis. Bonne[3] reported a case in which invasion of the pleura was definitely present. More recently Simon[4] was able to demonstrate the presence of metastasis to the brain in a case of alveolar cell carcinoma studied

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at autopsy. No metastasis, however, could be found in the bronchopulmonary or regional lymphnodes.

In view of the above findings Simon regards pulmonary adenomatosis as a slow growing, eventually metastasizing tumor, and concludes that pulmonary adenomatosis and alveolar cell carcinoma are the same. It has been suggested by many that the process may begin as hyperplasia stimulated perhaps by some specific agent, and then progressing to the stage of alveolar cell carcinoma.

The infectious nature of jaagsiekte has long been recognized and the work of Dungal, Gislason and Taylor[5] strongly support this view. A virus has been suggested by Cowdry,[6] Dungal,[7] and Bell,[8] but attempts to demonstrate an etiologic agent or transmit the disease to laboratory animals has been unsuccessful. Among sheep, on the other hand, the disease can be readily transmitted by housing normal sheep with diseased animals.

Although the disease had been prevalent among sheep in South Africa for many years, it was unknown in Iceland until 1934, at which time an epidemic began, with the subsequent destruction of a large number of animals. Dungal,[1] however, did not find any human cases in Iceland, not even among the shepherds, and concluded that man was apparently immune to this particular virus. More recently, however, Hildebrand[9] reported the case of a Montana ranch woman who died of pulmonary adenomatosis, after having been in prolonged contact with sheep one of which was subsequently proved to have succumbed to jaaksiekte.

In 1907 Helly[10] described an unusual lung tumor in a man, which resembled the condition found in the lung of sheep. Since that time more and more cases of this type of pulmonary involvement have been reported in the literature and there are unquestionably more cases which are not recognized or are unreported.

Bonne[3] suspected the possibility of a virus infection in the case of a Chinese male who died of this disease. Crane,[11] in 1945 reviewed the literature and was able to gather forty-three cases which he felt were true alveolar adenomatosis. Taft and Nickerson[12] reported two interesting cases in which the diagnosis was made from study of the autopsy material.

Abrahamson et al[13] describe an unusual case of this disease in which they felt the condition may have been caused by the use of thorotrast sixteen years previously. Recently Boucot et al[14] reported occurrence of focal pulmonary adenomatosis in a sixteen year old male, which was resected by lobectomy, without any evidence of

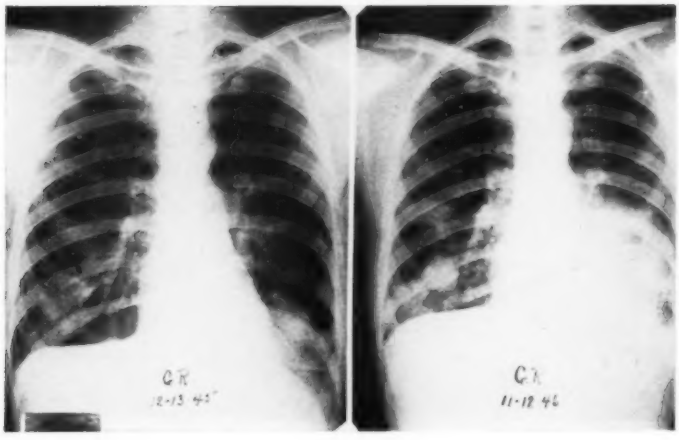

Fig. 1.

recurrence three years postoperatively. More recently McCoy[15] has reported an additional three cases. In two the diagnosis was made postmortem and in the third by study of the bronchial secretions obtained at bronchoscopic examination. Both the second and third patients were treated with nitrogen mustard without any benefit.

Watson and Smith[16] in a review of fifteen hundred and eighty-five malignant lung lesions treated at Memorial Hospital, were able to pick out thirty-three cases which they felt represented alveolar cell or bronchiolar cancer of the lung. The first case in their series was encountered in 1932 and eighteen cases were seen in the past two years and thirteen in the past year. In this particular group the average age was fifty-five years and only one-fourth were women.

The occurrence of this disease in three patients with scleroderma and pulmonary fibrosis is reported by Zetuchni et al.[17] The diagnosis in these patients was made as part of the postmortem findings.

The following cases are also being added to those already reported.

REPORT OF CASES

CASE 1.—G. R., a thirty-seven year old white male, was first seen at Abington Memorial Hospital in November 1946 because of

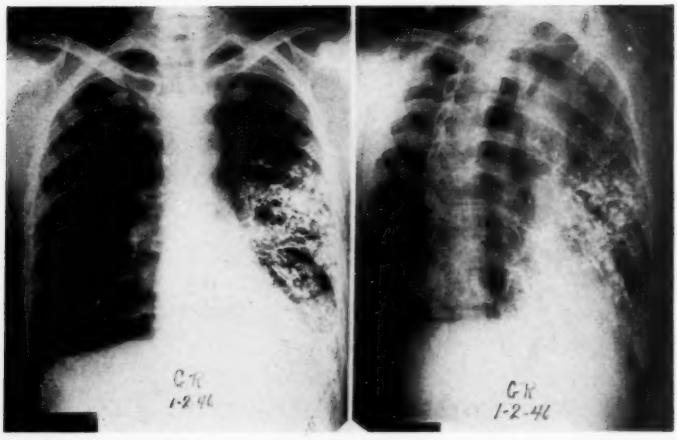

Fig. 2.

attacks of fever, 103-104°, associated with a grippy feeling, profuse persistent expectoration of frothy-like material, occasionally associated with blood-tinging. These episodes of fever had been going on for about two years. He had been x-rayed at Temple University Hospital on December 13, 1945 (Fig. 1, left) and bronchographic studies made on January 12, 1946 (Fig. 2) suggested the possibility of bronchiectasis of the left lower lobe. He was admitted to Abington Memorial Hospital at this time because of severe, painful and distressing cough and persistance of elevation of temperature of several weeks' duration.

X-ray studies of the chest on November 19, 1946 revealed in the posterior portion of the left chest, below the level of the seventh rib, a rounded shadow, flat against the posterior chest wall, and a small cavity just below the level of the eighth rib well posterior. On films made on November 26 this cavity was only vaguely seen, probably because it was filled with fluid with a minimum of air. The position of the cavity and the appearance of the shadow as a whole suggested it to be in the pleural cavity rather than in lung tissue (Fig. 1, right).

In view of the previous x-ray findings at Temple University Hospital and these studies made at Abington Hospital, it was felt that this patient had developed a lung abscess of the left lower lobe, secondary to his bronchiectasis.

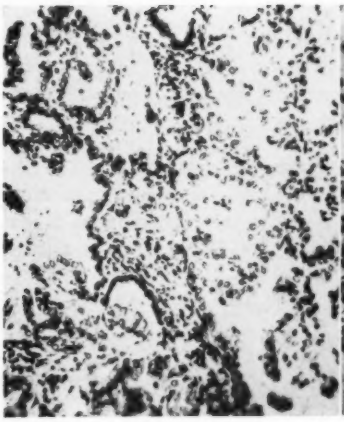

Fig. 3.

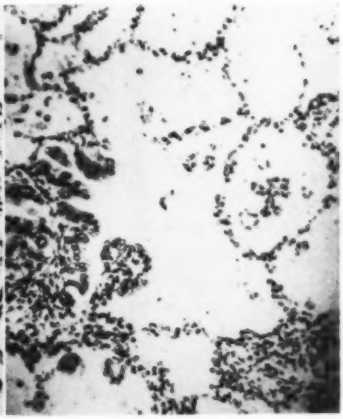

Fig. 4.

On repeated bronchoscopic aspirations and antibiotic therapy he showed remarkable clinical improvement. His cough disappeared and his temperature returned to and remained normal. He was discharged from the hospital entirely asymptomatic.

After being at home for about two weeks he again began to have recurrence of his previous episodes of chest pain, persistent hacky cough and elevation of temperature. Because of the severity of the patient's symptoms he was rehospitalized and was, on this occasion, admitted to the Pennsylvania Hospital, on December 13, 1946.

Another bronchoscopic examination was made at this time and some granulation tissue was taken for biopsy from the distal portion of the left main bronchus, which was found to be negative for any evidence of malignancy.

Because of the feeling that this patient had extensive damage of his left lower lobe from bronchiectasis it was believed that he might be benefited by lobectomy and Dr. John B. Flick, Sr. was asked to see him in consultation. Since it was suspected that he might have a localized effusion at the left base, a thoracentesis was made and 300 cc of serosanguineous, purulent material was aspirated. This material was sent to the laboratory for tumor cell study, at which time a peculiar type of tumor configuration was noted by one of the authors (A. Reynolds Crane) who also called attention to the re-

semblance of the histologic changes to those found in alveolar adenomatosis (Fig. 3).

On the basis of these findings a complete survey of this patient, including gastro-intestinal and genito-urinary studies, was made to rule out the possibility of some other primary focus, but none could be found.

In view of the suspicion at this time that we were most likely dealing with a case of alveolar adenomatosis, the entire pulmonary picture of this patient was reevaluated. The original films made at Temple University Hospital were compared with those subsequently made at Abington Memorial and Pennsylvania Hospitals. The seriological comparison of these films revealed a pattern of progressive changes which had not remained limited to the left lung field, but had spread to the right side, strengthening our suspicion that we were dealing with pulmonary adenomatosis.

In spite of the fact that we recognized that the span of life in this individual was limited, it was thought advisable to do a lobectomy, palliatively, in an attempt to give him relief from the severe sieges of agonizing pain localized in his left lower chest region, and lobectomy of the left lower lobe was performed by Dr. John B. Flick, Sr. on February 18, 1947, with uneventful recovery. Following lobectomy the patient was completely relieved of pain and had abatement of his cough, and he was discharged from the hospital on March 5, 1947.

The examination of the removed lobe showed typical histologic pattern of alveolar changes consistent with alveolar adenomatosis (Fig. 4).

Because of the progressive changes on the right side, noted on subsequent x-ray studies, it was thought advisable to try nitrogen mustard on this patient, and he received a full course without any apparent effect.

For about four months after lobectomy he got along fairly comfortably. Several months later, however, he again developed a persistent irritative cough, productive of frothy sputum. In addition he began to experience definite shortness of breath.

X-ray examination of the chest made at this time revealed marked progression of the involvement on the right side. In addition to marked infiltration involving the middle and lower lobe there was now definite evidence of abscess formation (Fig. 5).

The patient's dyspnea became so severe that he required oxygen constantly. The distressing cough was not relieved by sedation and

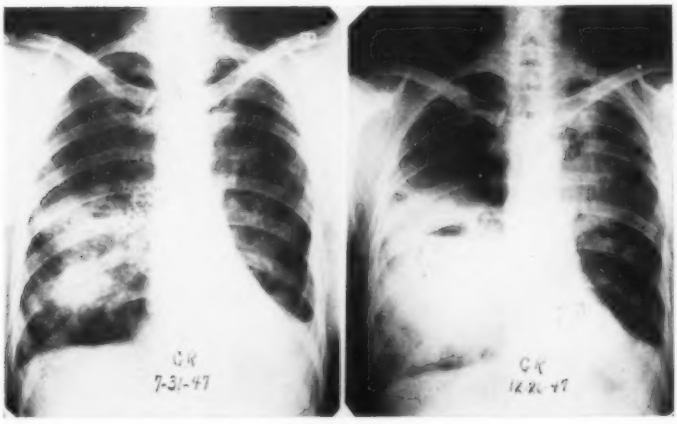

Fig. 5.

he went rapidly downhill and expired on February 15, 1948 at Abington Memorial Hospital. Unfortunately no postmortem examination could be obtained to conclusively prove that the lesion on the right side was of the same nature as that on the left.

CASE 2.—E. R., a white female 48 years of age, was admitted to the Pennsylvania Hospital on the service of Dr. Perry S. MacNeal on May 5, 1948, for review and further study of a pulmonary lesion which had not responded to treatment at home.

In July, 1947 she had an x-ray film of her chest made at the Burlington County Hospital during a routine survey, which revealed a slight degree of infiltration just above the left hilum. The possibility of tuberculosis was considered in spite of negative sputum examinations, and she was placed on bed-rest. For a time the lesion remained stationary but she began to have a more persistent cough and lost ten pounds in weight in six months.

On admission to the hospital x-ray studies on May 6, 1948 were reported by Dr. Paul Bishop:

"In our present examination there has been progressive increase in infiltration in this area, so that now we have a rounded, soft appearing, nodular lesion with indistinct margins, some 3 cm in diameter. The trunk shadow markings throughout the lung fields have gradually increased, so that now we have, scattered throughout both

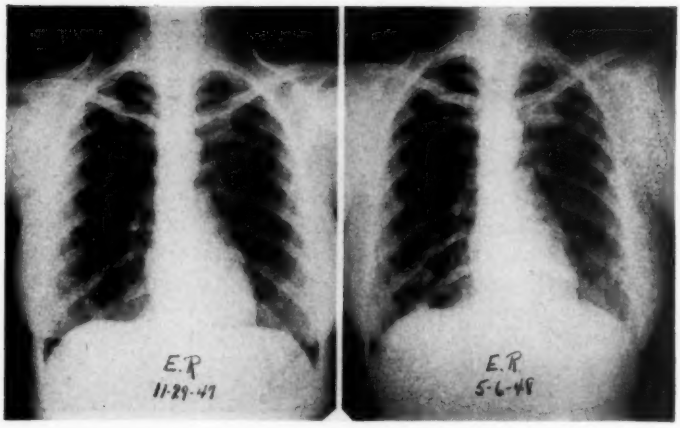

Fig. 6.

lungs, homogeneous on the two sides, multiple small areas of infiltration. I strongly doubt that this represents pulmonary tuberculosis, and would favor as my first opinion, that this is a manifestation of sarcoidosis. I also do not feel that a tumor, perhaps the oat cell type, with metastasis to the lungs, can be ruled out although this is highly improbable on the basis of the patient's clinical appearance and symptoms." (Fig. 6)

Laboratory studies showed: Histoplasmin—negative; coccidia—negative; blastomycin—negative; TB smear and culture of sputum—negative; blood studies—normal; Wassermann—negative; PPD first and second strength—negative.

A bronchoscopic examination was made on May 10, 1948 which revealed the motility of the larynx to be normal; the mucous membrane was quite inflamed. The mucous membrane of the trachea likewise was very fiery red and it showed some slight thickening. The bifurcation was not thickened, but again the inflammatory changes of the mucous membrane were rather marked. Careful examination of the right main bronchus revealed the caliber of the lumen to be normal throughout. The right upper lobe opening was normal. The middle and lower lobe openings showed no abnormal changes. There was a small amount of blood-stained secretion found on the right side. Careful examination was made of the left main bronchus and the mucous membrane here was more inflamed than on

the right, but there was no ulceration present anywhere. The lumen of the left main bronchus was of normal caliber throughout. An attempt was made to aspirate some secretions directly from the left upper lobe and this specimen was saved separately and sent to the laboratory for TB smear, study for fungi and study for tumor cells, in addition to routine bacteriological study.

From the x-ray studies and the bronchoscopic findings it was felt that we might be dealing here with an acid fast infection, and that the lesion in the left upper lobe might represent a tuberculoma. On the other hand the possibility of a new growth also was considered, although the slow progression of the lesion would favor its being acid fast in nature.

Studies for tuberculosis and fungi were negative. The cytologic study was reported as follows: "Smear stained by the Papanicolaou technique shows acidophilic granular debris in which are seen occasional erythrocytes. Occasional mucus-secreting cells are also present. Tumor cells are not identified."

She was discharged from the hospital on May 14, 1948 with clinical diagnosis of possible sarcoidosis; pulmonary tuberculosis? pulmonary malignancy?

On October 27, 1948 she was admitted to Jefferson Hospital with the chief complaint of productive cough for one and a half years, and dyspnea for three months. She stated that she had an x-ray examination in 1947 which was positive for tuberculosis. During the past three months she had shortness of breath accompanied by a feeling of pressure in her chest, even while at rest. She had had occasional hemoptysis and a weight loss of eight to ten pounds in six months.

Verbal report of carcinoma cells in secretions.

Dr. Frank F. Allbritten saw her in consultation on October 29, 1948 and he felt that, in view of the diffuse involvement of both lungs as demonstrated by x-ray examination, and positive secretions, etc., this was a case of adenomatosis or alveolar carcinoma of the lung. He did not recommend surgery but felt that nitrogen mustard therapy should be considered.

Some pleural fluid was sent to the Pennsylvania Hospital on November 4, 1948 which was positive for tumor cells, consistent with adenomatosis.

This patient died at Jefferson Hospital on November 27, 1948.

CASE 3.—E. C., a white female 55 years of age, was seen by Dr. David A. Cooper in consultation with Dr. Marcus W. Newcomb.

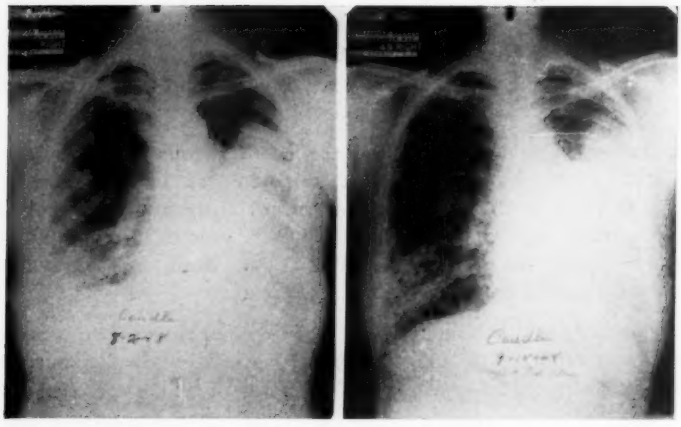

Fig. 7.

at Burlington County Tuberculosis Sanatorium, because of her puzzling pulmonary disease. She had had a persistent hacking cough productive of only small amounts of mucus.

The x-ray studies revealed bilateral pulmonary involvement, certainly suspicious of possible tuberculous infection.

Comparison of the x-ray films made on August 2, 1948 and September 18, 1948 revealed definite progression of the pulmonary lesions (Fig. 7).

Repeated sputum examinations for tubercle bacilli were negative. Gastric washings also were negative. In spite of the inability to demonstrate tubercle bacilli in repeated studies it was felt that this was the most likely possibility.

She failed rapidly and died. Postmortem examination revealed a most surprising finding in the histologic study of the lung tissue. The changes found in the alveolar structures were similar to those found in typical cases of alveolar adenomatosis.

CASE 4.—S. C., a forty-nine year old white female, was admitted to the Presbyterian Hospital on September 24, 1953, on the service of Dr. J. B. Donaldson, for further evaluation of her pulmonary disease, which had been previously considered as Boeck's sarcoid. She had been in good health until the winter of 1952 when she "caught a virus infection," at which time she developed a cough productive

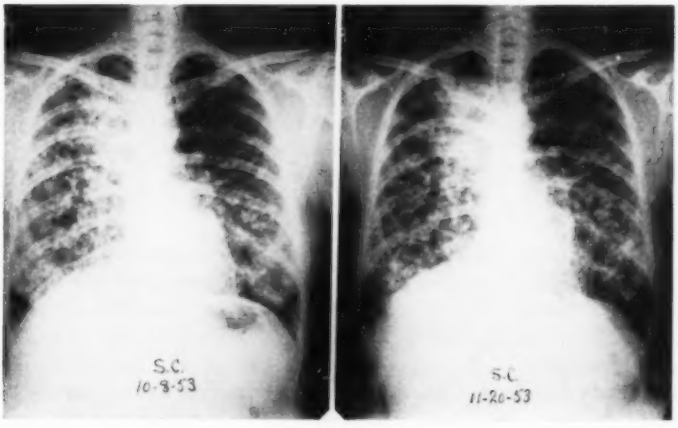

Fig. 8.

of mucoid, frothy-like sputum without any blood. The cough had persisted and occasionally was paroyxsmal in nature. Associated with this there had been increased shortness of breath, which was quite severe on admission to the hospital.

Physical examination revealed a definitely ill woman with apparent recent loss of weight and shortness of breath. The main physical findings centered in her chest which revealed diminished breath sounds bilaterally, with tubular breathing in both bases. Her temperature was 99,° pulse rate 100, respirations 24.

Blood studies showed — Hgb. 13.5 grams; RBC's 4,400,000; WBC's 6,400; Polys. 65%, Lymphs. 31%; Eos. 3%; Basos. 1%. Sed. rate—11.

Urinalysis was negative. Blood sugar 88; Urea Nitrogen 15, Serum proteins 6.0 gm, albumin 3.3; globulin 2.7, PPD negative.

Liver biopsy showed no histologic abnormalities.

X-ray examination of the chest, reported by Dr. Lame on October 8, 1953: "This study shows a generalized infiltration of the entire lung structures with a tendency toward some consolidation in the right upper third. The infiltration is of a spotty globular type. There are no demonstrable nodes, although some small ones may exist. There is no pleural effusion. The thoracic cage shows no lesion. Although this patient has had a diagnosis of sarcoid made, the lesion demonstrated here is somewhat atypical since there are no

demonstrable nodes around the hila and we understand there are no external nodes of abnormal nature. This lesion could well be carcinoma or other malignancy either primary in the lung or metatastic." (Fig. 8).

A bronchoscopic consultation was requested and in view of one of the authors' (W. A. L.) previous experience with another case, which presented somewhat similar lung pathology, the possibility of alveolar cell tumor was suggested.

A bronchoscopic examination was made on September 10, 1953 which revealed the epiglottis to be of normal contour; the mucous membrane was quite inflamed. Both cords appeared to move normally and there did not seem to be any true gross abnormality in these structures. The mucous membrane of the larynx appeared moderately inflamed. The lumen of the trachea was of normal caliber throughout, but the mucosal surface was very hemorrhagic and the mucosa itself was slightly thickened, but smooth. The bifurcation was slightly thickened. The lumen of the right main bronchus was normal in contour, but again the mucous membrane was fiery red. There was no evidence of any ulceration throughout the right main bronchus. There were some slightly blood-streaked, mucoid-like secretions aspirated from the right side. The findings on the left side were about the same as on the right. The amount of secretion was slightly less, but of the same nature. There was no endo-bronchial ulceration present that would fit in with the picture of tracheobronchial tuberculosis. The hemorrhagic changes and thickening of the mucosa were more indicative of a long-standing chronic type of infection. The secretions which were aspirated were sent to the laboratory for cytologic study, tuberculosis smear and guinea-pig inoculation bacteriological study, and study for fungi and coccidia. The bronchoscopic picture was very puzzling and the possibility of alveolar cell type of carcinoma was discussed with Dr. Custer, the director of laboratories.

Dr. Custer's report of the cell study of the bronchoscopically aspirated material was "Admixed among the many bronchial epithelial cells are cells encountered singly and in clusters, the majority showing excessive nuclear size with relatively small amount of cytoplasm and a considerable variability in size and shape. The nuclear chromatin of a number of these cells is coarse and irregular, some containing a conspicuous nucleolus. Pathologic diagnosis: Papanicolaou class IV-cells and cell clusters fairly conclusive of malignancy."

The other studies made on the bronchial secretions were negative.

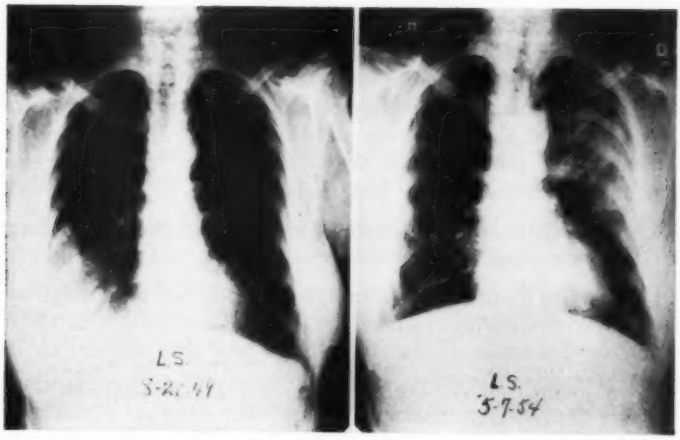

Fig. 9.

In view of the findings of the cytologic study and the clinical course of the patient, the persistent hacky cough, productive of frothy sputum led us to consider this patient as a fairly definite case of alveolar cell carcinoma.

She was started on a course of nitrogen mustard and received 6 mgm daily for four doses, following which she was discharged from the hospital, on October 18, 1953.

She was readmitted to the hospital on November 17, 1953, at which time she stated that she had not noted any improvement in her general condition; her cough had become more constant and the shortness of breath more severe. An x-ray film of the chest on November 20, 1953 was reported as showing no definite change since the examination of October 8, 1953. She was given another course of nitrogen mustard, 5.0 mgm for five doses, at daily intervals, and discharged from the hospital on November 21, 1953 with her general condition unchanged.

This patient subsequently died at home without a postmortem examination to conclusively establish this as a true case of alveolar cell carcinoma, which we felt it to be on the basis of the patient's symptoms, clinical course, x-ray findings and the cytologic findings on the bronchoscopically aspirated material.

CASE 5.—L. S., a sixty-five year old white female, was admitted to the Pennsylvania Hospital on August 18, 1949, with the chief complaint of cough, and weight loss. She gave the history that she had been well until six months previous, when she had the "grippe" followed by a persistent cough which had gotten progressively worse. She had been admitted to the Easton Hospital where x-ray studies revealed abnormal changes in the right lower lung field, and she was referred to Dr. John B. Flick, Sr. by Dr. Kincov for further evaluation and treatment of the lung lesion.

X-ray examination of the chest by Dr. Paul Bishop, on August 20, 1949 was reported as follows:

"Film and fluoroscopic examination shows that both diaphragms move freely and evenly. There is an area of infiltration in the posterior lateral portion of the right lower lobe that appears to show some segmental atelectasis. The trachea appears to be slightly over to the right. There is no definite mediastinal shift. The appearance is consistent with a new growth, although a chronic infectious process of long standing cannot be ruled out." (Fig. 9, left)

A bronchoscopic examination was made on August 22, 1949 which revealed the motility of the larynx to be normal. The lumen of the trachea was of normal caliber. The bifurcation was not thickened. The lumen of the entire right main bronchus was of normal caliber. The upper and middle lobe orifices were practically normal in appearance. The mucous membrane was only moderately inflamed. Careful examination of the right lower lobe bronchus revealed only one apparent abnormality, as far as could be seen by direct inspection, —the orifice of the medial division of the right lower lobe showed considerable inflammatory changes and appeared reduced in caliber, although there was no fungating tissue visible. An aspirator was passed into this subdivision and some bloody secretions were aspirated from it. The other subdivisions showed very little abnormality per se.

From these findings we felt that we were dealing with a peripheral lesion, and if it were a new growth it had not as yet extended into the larger visible bronchi. The history of the onset of symptoms certainly made one suspicious that it was a new growth rather than an infectious process.

The secretions which were aspirated bronchoscopically were negative for tubercle bacilli and negative for tumor cells.

Lobectomy, of the right lower lobe, was performed by Dr. John B. Flick, Sr. on August 27, 1949 and the histological examination of the lung tissue was reported as follows:

"The picture here is entirely consistent with pulmonary alveolar **adenomatosis** in which the alveoli have become lined by a mucous type of epithelium. Sections through proximal end of bronchi show no evidence of tumor. Lymph-nodes are negative for metastatic tumor. Diagnosis: Pulmonary Alveolar Adenomatosis, right lower lobe. Lymph-nodes show no tumor."

This patient made an uneventful recovery and was discharged from the hospital on September 8, 1949.

She was readmitted to Pennsylvania Hospital on April 30, 1954 with the history that two months prior to this admission she developed congestion in the left side of her chest. In addition she developed a persistent hacky cough, productive of clear mucus and without any blood.

She showed no improvement on treatment at home, and was referred to Dr. John B. Flick, Jr. for further evaluation of her pulmonary difficulty.

An x-ray examination of the chest by Dr. Paul Bishop showed "an extensive area of infiltration on the left side in the upper lobe. The lateral view shows this zone to be sharply demarcated by the fissure. It does not extend to the posterior chest wall nor to the anterior chest wall. It fades faintly towards the periphery but is homogeneous throughout. There is no evidence of cavitation. The appearance is consistent with a malignant infiltration, alveolar adenomatosis being the most probable. The lower lobe on the left side and entire lung on the right side appear normal." (Fig. 9, right)

A bronchoscopic examination was made on May 3, 1954 which revealed the lumen of the trachea to be of normal caliber, and there was no evidence of compression of the tracheal wall from the left side. The mucous membrane of the trachea, although inflamed, was smooth on the surface. Examination of the right main bronchus revealed the stump of the bronchus at the level where the right lower lobe was removed. The mucous membrane was moderately inflamed but there was no evidence of any endobronchial lesion. In examining the left side it was noted that there were considerable inflammatory changes in the mucosal surface with even some slight bleeding, but no ulceration. There was a considerable amount of thick, purulent material, slightly blood-stained, and frothy, found to be coming mainly from the left upper lobe orifice. There was no tissue within the lumen of the left upper lobe orifice.

Tumor cell study and tuberculosis study were negative.

In view of the previous findings in this patient it was thought that she had extension of the same disease to the left upper lobe, and this portion of the lung was removed on May 11, 1954, by Dr. John B. Flick, Jr.

The histological examination of the lung tissue was reported as follows:

"The gross specimen consists of left upper lobe, weighing 280 grams. Microscopic sections show the presence of papillary complexes of columnar mucous epithelium, diffusely infiltrating the pulmonary parenchyma in many places respecting the normal architecture of the lung and growing into alveoli in papillary prongs. Diagnosis: Diffuse mucinous adeno-carcinoma of the lung (pulmonary adenomatosis). No involvement of lymph-nodes."

In spite of her age this patient made a smooth recovery and was discharged from the hospital on May 22, 1954.

COMMENT

Although considerable new knowledge has been gained about pulmonary adenomatosis in the last decade there is still considerable questionable doubt as to the proper identity of this type of lesion from other forms of pulmonary tumors. As stated by Hutchinson,[18] "The subject is one in which much uncertainty and confusion still exist, for rarely have the histological investigations been sufficiently exhaustive to provide detailed information regarding the histogenesis of the neoplasm or its mode of extension in the lung, and it is lack of knowledge on these matters that is largely responsible for much of the present confusion." He feels that a great deal of this uncertainty in properly identifying this type of tumor could be eliminated if serial section studies were more frequenty utilized, in addition to the routine morbid anatomical and histological investigation.

In view of the bizarre unpredictable pattern which this type of lesion may manifest during its progression, very little help towards its identification can be gained from the patient's symptoms or clinical course of the disease during its early inception. Crane,[11] in a review of forty-three authentic cases of pulmonary adenomatosis found that this condition had been mistaken for the following types of pulmonary lesions, in the following order of frequency: pulmonary tuberculosis 10, pneumonia 7, tumor (unspecified type) 5, metastatic carcinoma 4, pulmonary carcinoma 3, fungus infection 3.

This further emphasizes the importance of the histologic evidence in the ultimate identification of this type of pulmonary growth. To this end the pathologist may be of inestimable aid, for without his

alertness in recognizing the characteristic pattern of pulmonary adenomatosis many of these patients would undoubtedly be classified under some other form of pulmonary pathology.

The first case here reported clearly illustrates this point. This patient was thought to have bronchiectasis of the left lower lobe, proven by bronchographic studies. When he subsequently developed changes in his left lower lobe suggestive of lung abscess it was thought to be secondary to his bronchiectasis. Although the material aspirated bronchoscopically did not show any tumor cells, the study of the material aspirated by thoracentesis did reveal tumor cells of a peculiar configuration, which aroused considerable curiosity as to its origin, leading to the suspicion and subsequent confirmation that we were dealing with pulmonary adenomatosis.

A survey of the early literature reveals that in the past the diagnosis of pulmonary adenomatosis was most frequently made at autopsy. Wood and Pierson[19] state that in all previously reported cases the diagnosis had been made postmortem. McCoy[15] found only one instance in the approximately seventy-five reported cases of alveolar cell tumors in which the diagnosis had been made correctly antemortem. Watson and Smith,[16] in their series of thirty-three cases, state that a clinical diagnosis of terminal bronchiolar carcinoma was made in twenty-two instances. This would indicate a progressive alertness to the existence of this clinical entity.

In the five cases here reported the diagnosis was established clinically in four. In one patient (Case 1)), although spread to the opposite lung was suspected, lobectomy was performed for relief of pain. In addition this patient received nitrogen mustard therapy without any apparent benefit, and died from contralateral spread of this disease.

In two patients, although the diagnosis was made antemortem, the pulmonary involvement was so wide-spread that no form of surgery was considered. One of these patients (Case 4), did receive two complete courses of nitrogen mustard therapy without any clinical improvement.

In one patient the diagnosis was made at autopsy.

Of unusual interest is Case 5. Here the diagnosis of pulmonary adenomatosis was made following lobectomy of the right lower lobe in 1949. This patient remained asymptomatic until recently when it was found she had involvement of the left upper lobe. This was resected on May 11, 1954 and the histological examination revealed the same type of histologic changes as had been found in the pre-

viously removed right lower lobe. This patient made an uneventful recovery and was discharged from the hospital on May 22, 1954.

Since alveolar adenomatosis may mimic the clinical signs, symptoms and x-ray findings of pneumonia, pulmonary tuberculosis, mycotic infections, Boeck's sarcoid, pneumoconiosis and metastatic carcinoma, one can appreciate the reason why it was not more readily recognized in the past before autopsy. The more recent reports, however, show an increased alertness to the existence and protean manifestations of this disease, so that many more cases of alveolar adenomatosis are diagnosed clinically.

CONCLUSIONS

In spite of its protean manifestations pulmonary adenomatosis is being recognized, clinically, much more frequently.

This may in part be due to better understanding of the histological characteristics of this disease.

Contrary to statistics on bronchiogenic carcinoma the incidence of alveolar adenomatosis is higher in women. Four of the five cases here reported were in women.

Although there is considerable histologic similarity between the disease found in man and that prevalent in sheep, no true relationship between these two diseases has been demonstrated.

255 S. 17TH ST.

We wish to express our thanks and appreciation to Dr. Perry S. MacNeal and Dr. Frank F. Allbritten, Jr. for permission to report Case 2; to Dr. David A. Cooper for Case 3; to Dr. R. Philip Custer and Dr. James B. Donaldson for Case 4, and to Dr. John B. Flick, Sr. and Dr. John B. Flick, Jr. for Case 5.

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DISCUSSION

DR. DONALD NICKERSON (Salem, Mass.): Mr. Chairman and members of the society: Tumors of the lung certainly are very important and interesting, and this particular syndrome which Dr. Lell has presented, and which I have enjoyed listening to, is one of the more interesting groups that has come across the desk of most pathologists in the past decade.

I think some of the confusion is slowly clearing. The syndrome does not resemble the usual carcinoma of the bronchial origin. Its peculiar distribution is unusual. Its origin is even more unusual. Recent evidence suggests that its origin may be from the mucous glands of the bronchial wall.

The terminology is a very confusing thing. Recently, reviewing the literature on this, I have encountered 31 different names applied to this condition, about half of them benign and half of them implying a malignancy.

Certainly, the multicentric origin of these tumors is most unusual. I think each case Dr. Lell presented had multicentral foci, either at the time or during the subsequent course of the patient. Certainly dissemination of the tumor through the lymphatics or through the alveoli could not, I believe, explain these observations.

The involvement of many lobes, or, in some instances, all lobes, in the non-metastasizing form of a disease is, I think, somewhat unique. The presence of similar nodules—in size—throughout the several lobes of the lung is also unique. There have been cases reported in which the nodules in the different lobes are identical in size, and there was not one large mass from which other masses might have been seated throughout the lungs.

The continuity between the bronchiolar or alveolar duct epithelium and the tumor has been demonstrated in some cases. In other cases—particularly, at least, in my knowledge, in our cases—we would find perfectly normal bronchiolar ducts surrounded by these tumor cells. I think this suggests a multicentric foci of origin of these tumors.

Dr. Lell has mentioned the relationship to animal tumors. It is of interest because of the histologic similarity between the animal tumors and, I think, the benign human tumors, in that in many instances of the benign form of the lesion, the cells are not ciliated, and they contain very large amounts of mucin. That is true in what little material I have seen in the similar tumors in sheep, in which cases the cells are not ciliated, and they contain rather large amounts of mucin.

As far as the morphology is concerned, I think the slides Dr. Lell showed you are truly representative. We look upon the benign lesion as of bronchiolar or alvelora origin. Our material was reviewed by Dr. J. Louis Bremer, who was kind enough to look at it for us, and he thought it was most interesting, because to an anatomist and histologist this is further evidence that the lining of the alveolus is epithelium, and under certain conditions can undergo metaplasia and perhaps neoplasia.

Of the malignant form, best called the bronchiolar carcinoma, of the 16 cases called carcinoma which we could review, there was certainly nothing to suggest that this arose from bronchial epithelium, as seen in the case of bronchogenic carcinoma.

The alveolar epithelium in these cases, as you have seen, is a tall, columnar epithelium filled with mucin, involving many alveoli, and producing frequently papillary fronds invaginating into the alveoli. I think the presence or absence of cilia is not terribly important. There is often associated a rather extensive degree of fibrosis, and chronic pneumonitis.

In our review of the material, we have been strongly in favor of making a diagnosis of this condition on the basis of exclusion. As Dr. Lell has suggested, a very thorough search for other foci of tumors must be made, and involvement of the bronchial mucosa would certainly put this in the form of primary bronchogenic carcinoma, and not alveolar carcinomatosis or adenomatosis.

We have not been fortunate enough to see a case ante mortem in which we could try ante mortem innoculation. Dr. Lell presented a case in which that was carried out, but other cases have been unsuccessfully transplanted to animals. It was our impression that peraphs these cases represent a viral type of lesion, because of the multicentric origin, and because of the frequency of multiple lesions developing subsequent to the first noticed lesion in the patient.

CHAIRMAN, DR. HOOVER: Dr. Lell, I do not believe that you mentioned the relationship of these particular patients to sheep or other animals. You must tell us whether there had been such a relationship.

This adds one more disease that we must keep in mind when we have an interesting case for which the diagnosis is unknown. You may not find the answer, but you should think of pulmonary alveolar adenomatosis.

Dr. Lell, will you conclude?

DR. WILLIAM A. LELL (Philadelphia, Pa.): There have been many attempts by some individuals, when they recognized they did have a natural case of true so-called alveolar adenomatosis, to inject laboratory animals, and in most cases they have been failures. Occasional reports in the literature give you a feeling that they are partly successful.

In the case of Simon, he felt definitely, from the circumstances and exposure of the patient which he described, that there was a possibility of a virus infection in his particular instance.

There is no question but that (at least it has been said) sheep cannot be innoculated through the transmission of blood samples from one sheep to another, and in that way getting the disease, and yet, on the other hand, if you take ill sheep and let them stay together, the others will eventually develop the disease in an epidemic form, if it is allowed to go too long. But actual laboratory studies are still very questionable as to the mode of transmission and the direct extension of the disease itself.

I just wish to say that there is so much confusion in the nature of this disease and its clinical course that one is thrown off balance. Unless you are alert to it, and unless you've seen a case or two, you certainly may mistake it for some other type of pulmonary disease.

I think this case of Dr. Custer's would probably have been reported as a possible metastatic tumor to the lung. The patient died at home. Unfortunately, no autopsy could be obtained.

The pathologist has to be careful in his diagnosis of material submitted to him for cytology. The first error might lie in the fact that he may not be able to demonstrate the cells because the proper sample of material has not been obtained. Then there is the cautiousness factor amongst some pathologists because of the medicolegal angle in saying the patient does have a tumor, when, later on, it may turn out that none exists. That has happened on some occasions. I think we have all had that experience.

LXXXII

RECENT TRENDS IN THE MANAGEMENT OF ESOPHAGEAL STRICTURES

CLAUDE C. CODY, III, M.D.

HOUSTON, TEXAS

The numerous reports in the literature concerning the handling of esophageal strictures, denote a gradually changing attitude in their management. It is true that techniques vary from early dilatation of acute esophageal lesions to those in which a stricture is actually permitted to form. Management of old strictures varies from conservative retrograde bouginage via gastrostomy to surgical resection. As is true in so many other situations, perhaps the answer is to be found somewhere between the two extremes.

The technique of a large university hospital prior to 1946, was previously discussed by the author. Since that time the cases have come, for the most part, from a large metropolitan area (Houston in Harris County, Texas) in a part of the country where there occur a large number of chemical esophageal burns, especially from lye. The most recent advances in management of esophageal strictures in this area are in the field of thoracic surgery. It is the purpose of this paper to classify such strictures in a workable manner and discuss the method in which they have been handled by our group. It is recognized that differences of opinion exist in this field, and this report merely presents a possible approach to the subject, one which has been handled successfully by our endoscopists.

ACUTE ESOPHAGEAL STRICTURES

Present literature indicates much acceptance of early dilation of acute esophageal burns after the method of Salzer and Von Bokay,[13, 17] that of early and daily bouginage of the acutely inflamed esophagus with soft bougies filled with mercury or lead shot. Such dilation is felt to retard the occurrence of subsequent strictures. According to Holt[8] "the incidence of stricture in the Harriett Lane Home has decreased from approximately 50% to less than 10%" since the introduction of this method.

From the Department of Otolaryngology Baylor University College of Medicine.

However, many of the cases so treated had mouth and pharyngeal burns and only "presumptive" evidence of an esophageal lesion. Obviously, some patients so treated would never have developed a stricture. A false impression of inhibition of stricture formation by early dilatation is thus obtained. No minor problem, when the esophagus is dilated at frequent intervals for a year or longer, is the follow-up care. In an area where many mouth burns are seen, it is increasingly apparent that some means of differentiating them from those associated with esophageal damage is necessary, in order to simplify the problems of after care.

Accordingly, late in 1946, a plan was devised for handling all acute lesions of the esophagus, whereby an attempt was made in every case to determine whether the burn was limited to the lips, mouth and pharynx, or whether it had extended to the hypopharynx, larynx and esophagus.

Emergency treatment consists of giving weak vinegar with copious amounts of water, diluted orange juice or a 2-per cent solution of acetic acid. This treatment is in all probability too late to prevent burn but it does no harm and, supplemented with small amounts of cream and olive oil, combats shock and supplies nourishment. As soon as the patient is able to take it, a regular diet is given. This may help to keep the lumen of the esophagus open and free of exudate. On the fourth or fifth day, when all evidence of shock has passed, careful examination of the mouth and the hypopharynx is made. If burns are found low in the hypopharynx and especially about the cricopharyngeus, no further instrumentation is done and the patient is immediately started on daily bouginage with mercury filled rubber dilators. If no burn is seen in the hypopharynx ,an esophagoscopy under general (ether) anesthesia is performed. The esophageal lumen is examined minutely for evidence of damage. Sometimes a whitish pseudomembrane is seen, sometimes a bead of granulation is seen, and sometimes the esophageal wall is quite hyperemic. In any event, it must be emphasized that as soon as the first evidence of esophageal damage is seen, the esophagoscope is withdrawn without looking farther into the esophagus, and daily dilation with mercury filled bougies is carried out. There are those who disagree with us on this early esophageal examination, but we feel that it is of real value. First, one can prove whether a prolonged regimen of dilation is really necessary or not; second, we can assess with more certainty the true value of early dilation on a burned esophageal mucosa. It is our feeling that an esophagoscopy properly and cautiously done under general anesthesia, and ceasing at the first evidence

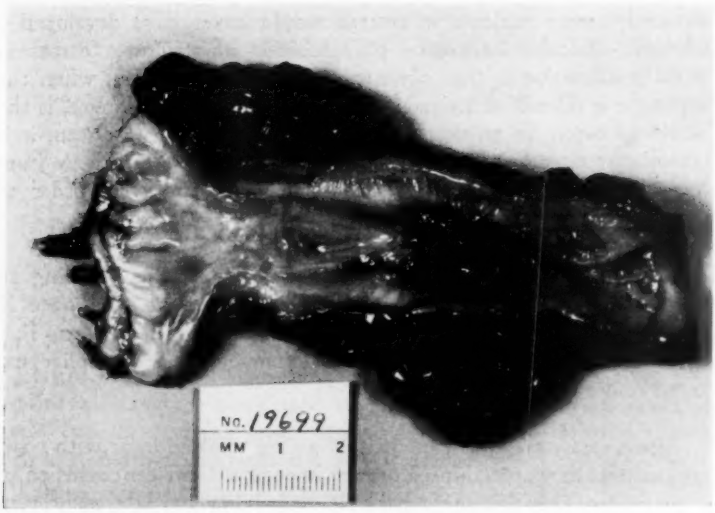

Fig. 1.—Resected specimen of case M. R. Note the very active inflammation and abscess formation seen in this specimen. This is the usual picture seen where there has been little or no preoperative esophageal dilation. Stenosis is almost complete.

of burn, need be no more hazardous than the use of soft mercury filled rubber bougies.

Intermediate Therapy (after 5 days). (a) After esophagoscopy on the fourth or fifth day has shown that true esophageal or hypopharyngeal injury exists, then daily bouginage is carried out for one month. Depending on the age of the patient, bougies increasing in size are passed by mouth, usually three or four at one sitting. These weighted bougies are allowed to be passed largely by their own weight until it is reasonably certain that they are well past the cardia of the stomach. The largest bougie passed on any one day is frequently left in place for twenty or thirty minutes. Occasionally, a febrile response follows bouginage, or there is bleeding from the esophagus. In such a case further dilatation may be delayed for one or two days and a smaller bougie resorted to again.

(b) After one month of daily bouginage and before the patient is discharged from the hospital, a second esophagoscopy under general anesthesia is carried out. The area under treatment is in-

spected and if it is healing, the instrument is passed below this area, and the lower esophagus thoroughly examined. Here it is important to note whether any active ulceration or granulation is present. Should this be the case, daily dilations must be resumed for another three or four weeks and then the esophagus is again examined. In treating all early esophageal burns, it has been found that best results can be obtained only when the patient remains in the hospital; and further, before the patient is discharged for out-patient care, the esophagus should be in a state of quiescence as seen through the esophagoscope.

It has proven both surprising and gratifying to note the number of cases of mouth burns following the accidental ingestion of lye or other caustic substance in which the esophagus was undamaged. In our series, for the years 1952 and 1953, there were 23 cases in which acute lip and mouth burns were present on admission. In only twelve of these, *only half*, was there any demonstrable esophageal pathology.

Patients who failed to reveal any esophageal burns have been discharged routinely, and although all were carefully instructed to return at once should they develop swallowing difficulty, none has returned. Conversely, in some cases of esophageal burns treated with early bouginage a stricture has developed.

(c) Follow-up consists of periodic dilation in the out-patient department at regular intervals. Some of the patients are discharged, given their own dilators and taught to pass them unaided. They are still required to return at regular intervals for re-evaluation, x-ray examinations and occasionally esophagoscopy.

LATE BURNED CASES WITH STRICTURE

Unfortunately, there are still far too many of well developed esophageal stricture, and the patient is not always at fault. It is a rather poor commentary on us that a patient discharged as cured may return some three to four weeks later with a well formed stricture which necessitates a long, tedious, and expensive regime. It is our responsibility to educate not only the public, but also the referring physician to the fact that early and intensive therapy is the treatment of choice in all esophageal burns.

This classification has been divided by us into a) short stricture, b) long or multiple strictures, and c) stricture (or patient) that responds poorly to prolonged dilation.

A) *Short Stricture.* Unforunately, this type of case rare. Initial therapy consists of treating the dehydration and malnutrition. Dur-

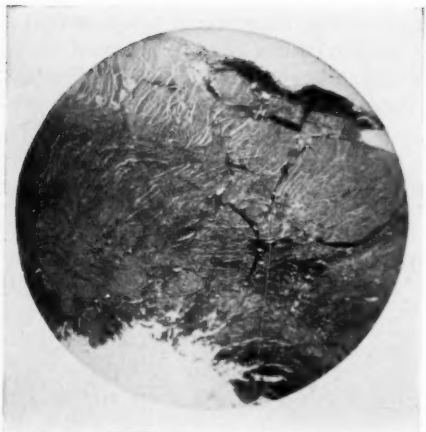

Fig. 2.—Photomicrograph, low power of case M. R.

ing this time preliminary x-ray studies are made and finally esophagoscopy is carried out.

The following example illustrates this type of case:

J. L., a white male, aged 13 years, was first seen on September 16, 1949. He was underweight and malnourished. He had been under the care of a psychiatrist as an emotionally unstable and nervous child and because of difficulty in eating associated with regurgitation and occasional chest pain, attributed to a nervous stomach. During the course of his examination, esophageal x-ray revealed a short benign stricture at the junction of the anterior and middle thirds of the esophagus. The boy's parents, on being questioned, recalled that the child had "licked" a spoon of lye when he was two years old, but they steadfastly maintained that he had not gotten enough to cause harm. After combatting dehydration for several days, an esophagoscopy was performed under general anesthesia. A firm ring-like stricture was encountered just at the junction of the upper and middle thirds of the esophagus. Scant granulations were present which bled easily on contact. Rigid dilators were used and the stricture was dilated with comparative ease. Subsequently the lumen was dilated to a French No. 34 under direct vision. At this stage, mercury dilation was started, a size up to French No. 26 being passed at weekly intervals for one month. The patient was then sent home (in an-

other city) after being taught to pass his own dilators. He is seen at regular intervals and about once a year when he experiences a little difficulty in passing his dilator, an esophagoscopy is performed and direct dilation carried out. This boy is now a strapping youth of 15 years. He has absolutely no evidence of dysphagia, although x-ray pictures still show a definite narrowing at the site of the old stricture. Certainly this youngster does not require surgery. Unfortunately, most cases of long standing stricture are not so easily handled.

B) *Long or Multiple Strictures.* This type of case usually presents the real headaches in management. Such patients frequently come in markedly dehydrated and show all the signs of starvation. Some of the younger children are seen in a critical state and may even die before adequate therapy can be administered. Again, early management requires control of dehydration and malnutrition. While this is being carried out with intravenous glucose and vitamin supplements, there is usually ample time for fluoroscopic examinations of the esophagus. Thin liquid barium sulfate or (in the case of infants) iodized poppy seed oil (4%) is found to be adequate and safe. After several days of preparation, an initial esophagoscopic examination, preferably under general anesthesia, is made to determine the tightness of the stricture. Under direct vision, a small flexible tipped bougie is used to detect the possible presence of any false pockets or pouches. We feel that it is important to note whether any reaction exists about the scar in the form of granulations or a pseudomembranous deposit. Vigorous dilation should be avoided or undertaken with caution if there is some reaction about the stricture.

It is rare that gastrostomy can be avoided in this group. When a small dilator will not pass readily from above into the stomach, gastrostomy is performed to initiate retrograde esophageal dilation. We have found that this actually shortens hospitalization, allowing the patient to return much sooner for follow-up treatments on an out-patient basis.

The type of gastrostomy we prefer is the Witzell type as this lends itself readily to the use of the Tucker[15] filiform bougies with less trauma to the stomach and the gastrostomy opening. It is fortunate when a string can be introduced into the stomach from above and picked up at the gastrostomy opening. Often this is impossible. Two weeks are then allowed for the margins of the gastrostomy wound to heal thoroughly. Usually a urologist then catherizes the esophageal opening from below. Distension of the stomach with sterile water allows better visualization with the pan-endoscope and

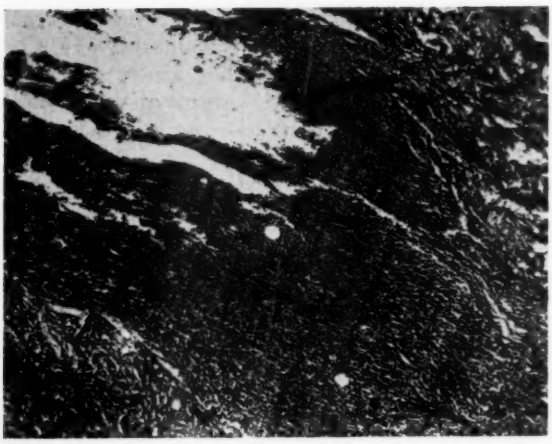

Fig. 3.—Photomicrograph, high power of case M. R. Note in this and the previous figure absence of esophageal epithelium, marked inflammatory reaction and polymorphonuclear infiltration in sub-epithelial layers. This high power section shows an actual abscess pocket in the stenosed wall of the esophagus.

the use of a small ureteral catheter. It has been interesting to note that this technique will be successful even when barium sulfate and the smallest dilator cannot be passed from above. It is our opinion that the esophageal stricture is never so tight at the distal as at the proximal end, since most of the effect of the chemical burn is in the region where the substance initially made contact with the esophageal wall. It is certainly true that catheterization of the esophagus from below is sometimes surprisingly easy when dilation from above is impossible. Once the ureteral catheter is passed through the esophagus from below, a string is introduced and retrograde dilation, after the fashion of Tucker, is commenced. Of course, this retrograde bouginage is done on an out-patient basis, and the patient takes all feedings by mouth. We have found that when such dilations have reached a calibre of French No. 36 to French No. 40 without undue forcing, mercury bouginage from above can be started. Many of the older patients are asked to purchase one of these dilators of a size that has been successfully passed and to perform their own dilations. Such a plan of therapy has proven effective and eliminates many of the inconveniences (and dangers) of the older direct method of dilation in which rigid dilators were used.

C) *Stricture or Patient Who Responds Poorly to Prolonged Dilations.* This group of patients has challenged the ingenuity of esophagoscopists for years and has been responsible for most of the failures in therapy. Both antegrade and retrograde dilations have usually failed because of lack of cooperation of the patient or his parents, a psychiatric personality, or a very long or multiple stricture in which there was a mechanical problem in dilatation. Such techniques as an indwelling catheter or a perforated filiform bougie, as described by Tucker, may give temporary results only to fail eventually. In this group of patients surgery may be the answer.

The recent advances in surgery of the esophagus are only partially due to the skill and technique of the surgeon. During the last ten to fifteen years interest in physiological surgery has begun to reap results. A better understanding of the mechanics and importance of fluid and electrolyte balance, along with the ready availability of blood for transfusions, have played an important role. Probably the greatest contribution to chest surgery has been made in the field of anesthesiology, where the proper understanding of pulmonary physiology, new technique of administration, and new agents permitting high concentration of oxygen have liberated the surgeon from the handicaps previously imposed on him. The availability of a longer operating time makes possible a more meticulous surgical technique. The maintenance of a clean tracheobronchial airway has all but eliminated postoperative pneumonia, as modern chemo- and antibiotic therapy have all but eliminated postoperative wound infection.

REPORT OF CASES

The following patients have been subjected to esophageal surgery since 1946.

CASE 1.—Mrs. M. R., white female, aged 24, had attempted suicide January 1, 1951, in a fit of acute depression. She was treated by a local physician with milk of magnesia and mineral oil. She was never able to take anything but milk and semi-solids, losing 146 pounds weight. A gastrostomy was done March 10, 1951. Retrograde dilations had been planned, but the patient never returned for this treatment. On March 8, 1952, the patient was admitted for complete study. X-ray examination revealed a stricture of the mid-esophagus about two inches in length. Just a trickle of barium would pass. On March 12, 1952, an esophagectomy with an esophagogastrostomy was performed. The operation was without event but the patient was a continued psychiatric problem and on March 25, 1952 left the hospital of her own accord. She has been seen at irregular

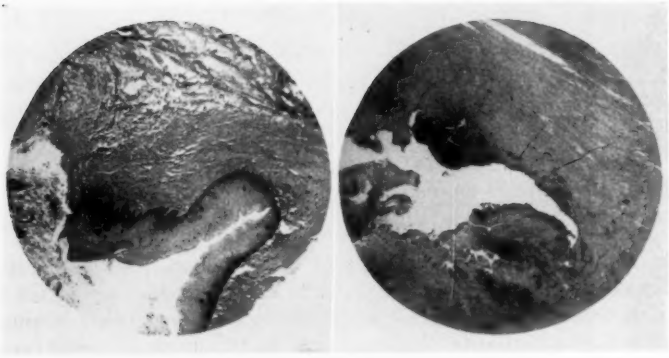

Fig. 4.—Case of J. A. T. Resected specimen with largely intact esophageal epithelium. Marked thickening of the esophageal wall with proliferation of fibrous connective tissue. Little active inflammation. This is the usual picture of the esophageal wall after a long series of dilations over a period of several years.

Fig. 5.—Case M. K. Please note extreme inflammatory reaction with loss of esophageal epithelium seen in this specimen where there has been little or no preoperative esophageal dilation. Actual abscess pockets may be seen on the esophageal wall.

intervals since and complains of difficulty in swallowing, and of bloating. She maintains a weight of 140 lbs. and apparently takes liquids and semi-solids better, although she can swallow solid foods. No recent x-ray has been obtained.

Comment: This patient is more fortunate than she realized. She can take nourishment with minimal difficulty. Unfortunately her mental status is so unstable that one wonders if she is worth salvaging. She would never have submitted to any course of prolonged therapy.

It is quite interesting to note the marked amount of infection of the esophageal wall in this case. Virtually, an abscess pocket existed in the stenotic lumen of the specimen removed. It has been found in this study that the more rigorously bouginage was pursued prior to surgery, the healthier the strictured portion of the esophagus became. Those cases with little or no bouginage presented the most reaction and infection. We feel this is an important finding.

CASE 2.—Mrs. J. A. T., white female, aged 30, had attempted suicide at 19 years of age. She had started retrograde dilations via gastrostomy in Philadelphia. The gastrostomy was later closed and

she had dilations irregularly up to October 27, 1948, when she was first seen by us. She was emotionally unstable, given to alcoholic bouts and attended "dope parties." She was examined initially because of obstruction which had been present for twenty-four hours. Esophagoscopy and dilation revealed a tight stricture just below the cricopharyngeus. This stricture was about 2 cm in length and a second stricture could be felt about 6 cm below the first stricture. Dilation to French No. 30 was accomplished by direct esophagoscopy and dilation. The patient would never return for follow-up care but would only come in when completely obstructed, usually after an alcoholic bout. Each time she was readily dilated to French No. 30. This situation prevailed until November of 1949. Eventually it was felt that esophageal surgery was the only recourse left and it was hoped that this would also help to stabilize the patient mentally. Accordingly, on November 14, 1949, a high esophagogastrostomy with resection of the rest of the esophagus was undertaken. A high thoracic anastomosis was made. Her postoperative course was quite uneventful. Some stricture developed at the site of the anastomosis, but this has yielded fairly well to periodic dilations. The patient has shown more emotional stability, but complains of fullness after eating and some difficulty in swallowing. Fluoroscopic study now shows that barium passes well into the stomach located in the left chest, but here there is considerable retention that still persists.

Comment: This young woman is certainly better off than before her surgery, but there is still room for improvement. Periodic dilations are still needed. On the whole this procedure has been considered successful.

The esophageal mucosa is healed. There is marked thickening and scarring of the esophageal wall, but little if any active infection is to be seen at the site of the stricture. Such a case would have continued to do well with periodic dilations had proper co-operation been possible. We believe that periodic dilations reduce reaction and help bring about a more rapid and effective dilation. This young woman is now carrying on a gainful occupation as a business manager in a beauty shop and requires only occasional dilations.

CASE 3.—M. K., colored female, aged 24, attempted suicide by lye ingestion August 4, 1949. She was seen in the emergency room where immediate therapy of dilute acetic acid was instituted. On the fourth day after the burn, a direct esophagoscopy under general anesthesia was carried out.

A whitish pseudomembrane was noted at the junction of the upper and middle thirds of the esophagus. The instrument was im-

Fig. 6.—Case S. D. Specimen where dilation had been carried out but stricture continued to develop. Very little inflammatory reaction is present, but marked thickening of the esophageal wall with fibrous connective tissue has developed. This child was a keloid producer.

Fig. 7.—Case of J. L. Specimen of resected esophagus. Sporadic preoperative esophageal dilation. There is some loss of epithelium associated with sub-epithelial inflammation indicating possible granulation tissue. However, much fibrous connective tissue is present in the esophageal wall. Possibly more vigorous dilation would have been effective in this case.

mediately withdrawn and mercury bouginage was instituted. The patient was then transferred to the out-patient service for regular dilation, but she did not return. Five weeks later, she was brought into the clinic almost completely obstructed. She was then initially hydrated, during which time x-ray studies showed a well formed stricture at the junction of the anterior and middle thirds of the esophagus. Esophagoscopy was then done with the purpose of investigating the stricture directly. One of the residents was overenthusiastic in attempting to dilate the stricture and perforated the esophagus. An immediate thoracotomy with drainage was instituted. It was felt that such an unco-operative patient would be difficult to handle under the necessarily long course of retrograde bouginage via gastrostomy.

Accordingly, on November 14, 1949, an esophago-gastrostomy was done, removing the lower two-thirds of the esophagus. The stomach was brought up into the left chest. This woman's postoperative course has been most uneventful of all cases studied. She has been seen only occasionally in the clinic since her operation and only on one occasion has she required any dilation.

Comment: This patient has been the most gratifying of our series following surgery. Her only complaint is occasional bloating of the stomach and fullness of the left chest. The removed specimen in this case again showed the extreme reaction and infection seen in the stenotic portion of the esophagus where little if any dilation had been performed previous to surgery. Esophageal mucosa was denuded and the esophageal wall was filled with multiple small abscesses.

CASE 4.—S. D., a colored male, aged 3, was admitted to the emergency room May 17, 1953, following ingestion of a spoonful of lye. Admission to the hospital was first refused by the mother. Then on May 20, 1953, the child was admitted to the hospital as an emergency because he was able to take only water by mouth. Burns were present on the lips, gums, tongue and pharynx. The child was given intravenous fluids and vitamin supplements. X-ray studies of the esophagus showed no pathological change. May 25, 1953, esophagoscopy showed a rather diffuse esophagitis but no definite area of membrane. Mercury bouginage was instituted as a daily procedure. Esophagoscopy June 2, 1953, under general anesthesia revealed a three mm area of granulation tissue just at the junction of the first and middle thirds of the esophagus. Continued mercury bouginage was maintained rather sporadically in the out-patient department until November 16, 1953. Repeated esophagoscopies showed a persistent tight stricture 4 cm in length in the mid-portion of the esophagus. At this time only a 4 mm Jackson esophagoscope could be inserted into the stricture with difficulty. In this case, because of the extreme difficulty in follow-up, surgical resection of the esophagus rather than gastrostomy was decided on.

December 17, 1953, a high resection of the esophagus with an esophagogastrostomy was performed. From this date to February 8, 1954, the child would only take liquids, and vomited frequently. He ran a very septic course. On February 8, esophagoscopy revealed an extremely rigid scar at the site of the anastamosis and this would admit only a French No. 8 rigid dilator with difficulty.

Weekly esophagoscopies gave the same results. On March 3, a revision of the anastomosis was carried out after first doing a tracheotomy. A massive ring of scar tissue, almost like keloid, was removed at the anastomotic site. The child has had gentle mercury bouginage beginning two weeks postoperatively. He still tolerates only liquids and semisolids and is frequently beset with a septic fever. To date he is still in the hospital and No. 17 and No. 16 mercury dilators can be passed by mouth.

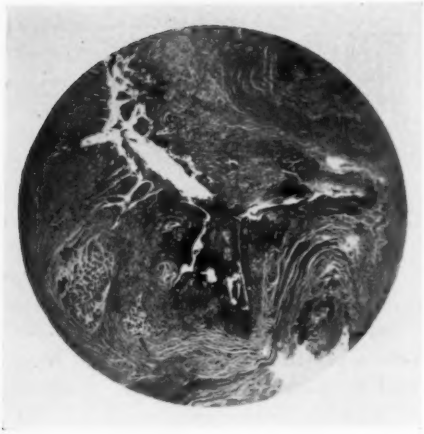

Fig. 8.—Case M. S. Again note the considerable inflammation about the esophageal lumen. Dilations were sporadic and irregular. Deep to the inflammatory area is the thickened esophageal wall largely replaced by fibrous connective tissue

Comment: Certainly gastrostomy and retrograde bouginage would have been better. Perhaps the tendency to form scar tissue played a big role here, yet the hypotonic stomach has plagued the youngster with almost continual digestive disturbances. The only possible advantage in this case is that the child has been forced to remain continuously in the hospital under constant observation.

From the above evidence, it is now felt that even with a long stricture such as this, retrograde dilations may have been of advantage in reducing reaction and infection in the long stenotic area of the esophagus. It would seem that conservative therapy is the treatment of choice in children.

Case 5.—T. L., Mexican female, aged 2, was seen on March 23, 1946, three months after ingesting lye. She was only able to swallow liquids. X-ray studies and esophagoscopy revealed a three cm burn at the junction of the upper and middle third of the esophagus. The smallest dilator could not be passed from above. Gastrostomy and retrograde dilation were started and continued until November 1949. Dilation could rarely be maintained above No. 26 French and the child was not returned frequently to the clinic.

November 21, 1949, an esophagogastrostomy was performed. Postoperatively some stenosis developed at the anastomotic site. To date liquids go down well, but solids tend to be caught at the stricture site. On two occasions in 1952 foreign matter has had to be removed via esophagoscopy from the anastomotic site. On March 1954, esophagoscopy revealed a firm irregular stricture at the esophagogastric junction which would just admit a five mm esophagoscope.

Comment: This case has had a fair result. She has gained weight and carries out most normal activities. She has learned to chew her food carefully and eat slowly. She occasionally vomits if she eats too fast or too excessively. She was unable to maintain adequate dilation by a prolonged course of retrograde bouginage. On several occasions indwelling rubber catheters were left for 48 hours within the stricture site, but without lasting benefit.

CASE 6.—M. L., a 34 year old white male, drank a half cup of lye on July 18, 1950. He had emergency treatment in the out-patient department at that time, but signed his own release. He was somewhat mentally disturbed. He returned August 8, 1950, able to swallow only liquids with difficulty. He had lost 30 pounds of weight. Barium studies revealed a complete obstruction in the middle third of the esophagus. Esophagoscopy revealed a dense stricture through which the smallest dilator would not pass. After combating the dehydration, a gastrostomy was performed August 16, 1950. The patient was handled via retrograde bouginage, but because of his disturbed mental status he would frequently have severe episodes of depression, especially following the dilations. After due consultation, it was felt that surgery might help to convert this man into a useful citizen. On March 5, 1951, an esophago-gastrostomy and resection of the stenotic lower esophagus were performed. The patient was extremely disturbed postoperatively and required heavy sedation. On March 14, 1951, an empyema of the right chest was drained. On March 19, 1951, a jejunostomy was performed when it was felt that a definite fistula had developed in the right chest. On March 24, 1951, the patient died suddenly.

Comment: This must be considered a death as the result of surgery. The patient was completely unco-operative and mentally confused. Yet had the surgical technique been faultless the patient might possibly have returned to some use in society. (See case No. 2.) These patients are not good risks at best and every effort should be made to help them adjust to their place in society.

CASE 7.—J. L., a colored female, aged 24, drank lye in a suicide attempt. She was given emergency treatment in the out-patient

department on January 3, 1946. Esophagoscopy six days later revealed some membranous formation in the mid-portion of the esophagus and immediate bouginage with soft mercury dilators was undertaken. In spite of this therapy, increasing obstruction developed and eventually the stricture required rigid dilation each time. On April 6, 1946, a gastrostomy was performed and retrograde dilation undertaken. It was quite difficult to maintain the esophageal lumen above a French No. 16 or No. 18 calibre. On consultation resection was advised and on November 18, 1946, resection of the scar and an esophagogastrostomy was performed. This patient has done well to date. Slight dysphagia exists. Minimal scarring at the site of anastomosis is present.

Comment: Note the thick, well-formed esophageal mucosa and absence of infection in the esophageal wall. Considerable dense scar tissue without inflammation is to be seen in the esophageal wall. Again it would seem that the older patient adjusts to this major surgery with less difficulty than the young child.

CASE 8.—R. B., white female, aged 21, had attempted suicide by swallowing lye in May of 1948. Emergency treatment was administered in the clinic and the patient refused any further treatment. Some six weeks later she returned unable to swallow anything except a scant amount of liquids. She had lost weight and was dehydrated. X-ray studies and esophagoscopy revealed a tight stricture at about the mid-esophagus. Gastrostomy and retrograde bouginage were begun about July of 1948. This method of dilation was continued irregularly for two years, seldom obtaining a dilation above 18 or 20 French. From 1950 to 1953 the patient returned very irregularly for dilations, taking liquids or semisolids by mouth. It was finally felt that inasmuch as only a No. 18 French Tucker bougie could be passed with difficulty, surgery could certainly offer some improvement On May 28, 1953, resection of the stricture and the lower esophagus with an esophagogastrostomy was done. The patient has since that time returned to a fairly normal life although still subject to depression. She has multiple complaints, but eats normally and has gained weight.

Comment: An adequate trial of conservative management had certainly been given this patient. Again the specimen in this case showed ulceration and abscessed pockets where regular and periodic dilatations had not been maintained. This case also demonstrates that adults seem better adjusted to major esophageal surgery.

CASE 9.—M. S., Mexican female, aged 2, was first seen in March of 1947 for an acute lye burn. Mercury bouginage was instituted in

the usual fashion, yet a tight stricture developed. A gastrostomy and retrograde dilation were carried out from 1947 to May 1952. Very little dilation could be maintained by retrograde bouginage, a French 16 or 18 bougie being about the largest admitted. On May 29, 1952, esophageal surgery with resection of the stricture and an esophagogastrostomy was carried out. The child had a stormy postoperative course but at this time is gaining weight and has only a minimum of digestive complaints.

SUMMARY

In our experience, there is considerable evidence in favor of keeping the damaged esophagus, if this is at all possible. In this we disagree with some other authors (Burford, T. H., et al[2]). The major problem after the initial surgical procedure is to prevent further stricture at the site of anastomosis. Almost all of the patients have digestive disturbances varying from slight to quite severe. The author has shown in an earlier publication that such cases can be handled conservatively in most instances. Certainly the substitution of one stricture for another of surgical origin cannot be considered a successful procedure. Recently, mercury bouginage has been used after esophagogastrostomy in an effort to prevent stenosis. Results are inconclusive. Many thoracic surgeons now feel that the postoperative surgical strictures which develop are the result of regurgitation of stomach contents from the atonic stomach into the site of anastomosis rather than the result of surgical trauma.

We feel firmly that in all cases of chemical burns of the mouth and pharynx esophagoscopy should be carefully done on about the fourth day after the burn. If done cautiously this should cause no harm and it reduces almost in half those cases which would otherwise require mercury bouginage for a year or more. Where the follow-up problem is already tremendous, this has a distinct advantage. It will also tend to place in a more correct light the reported good results of early mercury bouginage.

Finally, there will always be cases that do not lend themselves well to dilation. We feel that these patients after consultation, probably should be considered candidates for surgery. Certainly as technique develops, fewer sequelae after surgery will ensue. We have had no experience with ACTH and Cortisone, although these appear to have some merit. Obviously, every case must be handled in the light of its own findings. However, we feel that if the continuity of the esophagus can be established and maintained this has a great advantage over any surgical correction, and one should resort to surgery only when it is obvious that conservative measures will not suc-

ceed. We further believe from this study that conscientious effort at dilation will, in most instances, prove successful in that infection and inflammatory reaction are greatly reduced.

Sporadic or irregular dilation reduces the chances of success and allows infection and further stenosis to develop. Many of these patients need psychiatric care and any patient, he must be treated as a whole in order to get satisfactory results.

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DISCUSSION

DR. BENEDICT (Boston, Mass.): Mr. President, members and guests: In suspected lye strictures of the esophagus, I have felt that one of the first things to do was to have the patient swallow a thread, and then we always have a lumen in the esophagus to the stomach, and we can carry out bouginage, if necessary.

Then I also think it is very important to follow the situation by x-ray examination of the esophagus, so that we can see if a stricture is developing or if it is not developing.

As far as gastrostomy goes, I rarely use a gastrostomy, because I do the bouginage through the mouth without gastrostomy and avoid gastrostomy if possible.

DR. DONALD S. BOLSTAD (Detroit, Mich.): I would like first to congratulate Dr. Cody on his fine presentation of a long discussed problem. Secondly, I wish to compliment Dr. Benedict for his comments regarding the swallowed string without gastrostomy. For the past seven years I have been in the practice of dilating by the Plummer method over the swallowed string without gastrostomy.

I believe this is advantageous from two standpoints. First, it avoids the drawing of a retrograde dilator from the stomach into the esophagus and thus overcomes a tendency to cause irritation from gastric secretions. Secondly, in elderly patient at least, by eliminating the possibility of feeding through the gastrostomy tube one is able to keep in better contact with those who fear or resent the experience of dilatation to the extent they fail to return. There are, also, a few parents who prefer occasional feeding per gastrostomy to bringing their youngster for regular dilatations.

DR. WALTER BOYD HOOVER (Boston, Mass.): I think Dr. Cody has pointed out the poor cooperation many of these patients give. That is one of the great difficulties in the successful treatment of these patients.

A person who is despondent, who is likely to commit suicide, or has tried, is a very unreliable patient. When you have a patient who cannot swallow anything, if esophagoscopy has been done and nothing can be passed through, then a gastrostomy must be done—there is no choice.

I had one patient who, by mistake, drank varnish remover instead of liquor. He came to me when he could no longer swallow any fluid. I esophagoscoped him, and was afraid to attempt dilatation because of the ulceration and granulation. I withdrew my esophagoscope and told him he would have to have a gastrostomy. He said, "Doc, you'll have to try again. I'd just as soon die as have one of those."

I said, "Well, you may die." Two days later I attempted unsuccessfully to pass a dilator, and mediastinitis developed. Then he was willing to have proper treatment, and the results were quite successful. That was ten years ago.

I had a letter from him this week; he is in a hospital now, and cannot swallow. He had refused to come in for follow-up examinations.

I am sure that we will have to individualize treatment. We will have to do what we can with the cooperation offered us. Good results will never be obtained consistently in this type of patient.

DR. HOWARD W. D. MCCART (Toronto, Canada): I would like to mention one other method of treatment in the management of Esophageal Strictures. Mr. Victor Negus described this procedure in the Journal of Otology and Laryngology, January, 1953, where the construction of an antethoracic esophagus had been contemplated. Three hollow plastic bobbins were made the smallest 2" long, maximum diameter ½", medium size 2" long maximum diameter ¾", the largest 2¼" long maximum diameter ⅞". An eyelet at the upper end allows the bobbin to be tied to a continuous thread which passes lengthways through the centre.

Tension is applied to the thread emerging from the gastrostomy by a strong elastic band anchored to the abdominal wall. The average time for a bobbin to pass through a stricture is 5-8 days.

Recently an old man was referred to us from the Department of Surgery. He was in the habit of keeping a bottle of whiskey in the garage as it was against his wife's principles to allow whiskey in the house. One day he reached for the whiskey and in error drank from a bottle containing battery fluid.

Following this no attempt at bouginage was carried out but instead a gastrostomy was performed. Some 18 months later he was referred to our Department. A thread finally worked its way through the esophagus and was brought through the gastrostomy opening; following this esophagoscopy was carried out revealing three strictures measuring about 10 mm in diameter at the level of the cricoid, level of the left bronchus and at the hiatus. Retrograde bouginage was carried out for six weeks without result, the same was true for an indwelling tube. Finally, the above mentioned plastic bobbins were tried, the first came through in 24 hours, the second in three days, the third in about 10 days. It is now about three months since this procedure was carried out—the patient is swallowing all types of food and can pass a 28 mm Hurst mercury bougie without any difficulty, the gastrostomy opening is being allowed to remain patent for another three months.

DR. HOLINGER: I would like to compliment Dr. Cody on his paper, but I would like to speak a word *for* gastrostomy rather than against it. The gastrostomy is used for the purpose of prograde rather than retrograde dilatation, as suggested by Barretto and Botelho of Sao Paulo. They use a Plummer bougie over a string brought out through the gastrostomy and held firmly during the dilatation. This type of dilatation from above rather than below, has been the most rapid and most satisfactory technic of dilatation of tight, old strictures that we have found.

The recurrence of a stricture following surgical resection of strictures was mentioned by Dr. Cody. I believe that jejunal-esophageal anastomosis, rather than a resection of the stricture and an esophagogastric anastomosis, is more satisfactory. Why remove the esophagus at all? Bringing a loop of intestine above the point of stricture apparently prevents the gastric juice from being regurgitated to reform a stricture. This technic seems to be more satisfactory than resection of the stricture itself.

DR. F. JOHNSON PUTNEY (Philadelphia, Pa.): I would just like to add a word to what Dr. Holinger has said, and also to second his remarks about the use of the string.

This can be used without a gastrostomy, even in small children. If one has a string through a stricture, which is not too tight, bougies can be passed over the string with fast dilatation. I do not know that a gastrostomy is absolutely necessary in this type of treatment unless the child is not getting sufficient nourishment.

DR. MURDOCK EQUEN (Atlanta): Dr. Cody's paper has been most interesting. Lye strictures are becoming rarities today because it is just as cheap to buy soap as to attempt to make it and lye is not being used for cleansing purposes. At the same time, there will always be an occasional lye stricture as well as strictures due to other causes and we must be always ready to treat them.

When difficulty is experienced in getting a child to swallow a string, we pass a cone shaped bit of steel on the end of a semi-flexible wire under fluoroscopic guidance through the esophageal stricture into the stomach. Then an Equen magnet is passed into the stomach through the gastrostomy. The magnet is withdrawn together with the steel cone and a string is tied to the cone. The cone and string are drawn back up the esophagus through the mouth, thus permitting retrogade dilatations.

DR. CLAUDE C. CODY, III (Houston, Texas): I wish to thank all of the discussers of this paper. I indicated beforehand there was considerable difference of opinion in this subject.

The length of my paper prevents going into extreme detail, but we do not do routine gastrostomy procedures except when we are unable readily to enter the stomach from above. Usually it is the long stricture or the multiple strictures in which we have attempted to enter the stomach from above, with or without a string, that we have resorted to gastrostomy rather than proceed with a long and extremely trying procedure. In our hands it has shortened the overall treatment; further, it considerably shortens the period of hospitalization and helps bring about more rapid nourishment and rehabilitation of the patient, particularly a small child. In summary, I would say that our group has a little better success in employing the gastrostomy (Witzel type) where it has been quite difficult to enter the stomach from above.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, February 1, 1954

THE PRESIDENT, DR. ROLAND RUSSELL, IN THE CHAIR

Surgery in Carcinoma of the Hypopharynx

IRWIN D. HORWITZ, M.D.

ARTHUR LOEWY, M.D.

BURTON J. SOBOROFF, M.D.

AND

MAURICE F. SNITMAN, M.D.

CHICAGO, ILL.

(Complete paper appears on page 993)

Early Prosthetic Closure Following Maxillary Resection

STANLEY H. BEAR, M.D., MAJOR M.C. U.S.A.F.

AND

ROLAND A. KOWAL, M.D., D.D.S.

CHICAGO, ILL.

(Complete paper appears on page 1024)

DISCUSSION

DR. MAURICE F. SNITMAN: If one were to exclude the beneficial effects outlined by Dr. Kowal on the value of the prosthesis, and apply it solely for the purposes of intracavity radium application, this prosthesis would be well worth the effort expended in its construction. Following radical surgery of the maxilla for neoplastic disease, the intracavitary radium application is indicated. This procedure is simplified by this prosthetic mold, in maintaining in position the radium needles or radon seeds.

Electrodiagnostic-Therapeutic Modalities in Facial Nerve Paralysis

ARTHUR A. RODRIQUEZ, M.D.
AND
EMANUEL M. SKOLNIK, M.D.
CHICAGO, ILL.

(Complete paper appears on page 1015)

Tracheotomy in Infancy

PAUL H. HOLINGER, M.D.
KENNETH C. JOHNSON, M.D.
JOHN A. BIGLER, M.D.
AND
FILMORE SCHILLER, M.D.

(Abstract)

The incidence of tracheotomy in infants under one year of age is increasing due to broadening of indications, greater acceptance of tracheotomy as an adjunct to other therapy and a lower mortality through improvements in surgical technique and in postoperative care of tracheotomized infants. Tracheotomy for inflammatory lesions is decreasing, but the incidence of tracheotomy for congenital and miscellaneous lesions is increasing. Of 62 infants under one year of age who required tracheotomy, congenital lesions were considered the cause of the respiratory obstruction in 32 cases (15 males and 17 females) and acquired lesions in 30 (21 males and 9 females). In 8 of the 30 infants in the "acquired" group, congenital anomalies were indirectly responsible for the tracheotomy since it was necessitated following extensive surgical procedures performed in an attempt to correct gross anomalies of organs other than those of the respiratory system. There were 17 deaths in the series of 62 cases. Eight occurred in the miscellaneous group, principally those operated upon for gross anomalies of other organs, 3 in the group of 17 congenital laryngeal anomalies, 4 in the group of 18 inflammatory lesions and 2 in the group of 8 neoplasms. Tracheotomy is generally looked upon as an emergency, life-saving procedure. However, in most cases it can be a planned, careful operation, preceded by the

establishment of an airway to avoid cerebral and surgical trauma. The post-tracheotomy period is the critical period during infancy.

DISCUSSION

DR. A. H. ANDREWS: There are a number of aspects of the subject presented by Dr. Holinger upon which one might dwell for the purpose of emphasis. One youngster he mentioned was interesting. The child was taken to a hospital with the story that he had been playing alone in a room when the mother heard him choke and cough. Examination by a resident demonstrated no symptoms or findings, and the mother was told to bring him back next morning. Again there were no symptoms or findings and the mother was told that he was all right. She became angry and before an x-ray could be taken she left the hospital.

Some six weeks later the child was admitted to Children's Memorial Hospital with the tentative diagnosis of acute laryngotracheal bronchitis, with classical symptoms. Tracheotomy was needed. At the time of the bronchoscopy I saw a shiny foreign body in the larynx, took hold of it with forceps and, with gentle traction, withdrew it. It was the keeper of a safety pin. The child stopped breathing and a small bronchoscope was passed alongside the pin, the airway established, and a tracheotomy was done disregarding the safety pin. Next morning the trachea was exposed and the pin was grasped by the posterior limb and pulled up into the tracheotomy wound, perforating the anterior wall of the trachea with the anterior branch. This was cut off with wire cutters and the safety pin rotated out. The child had a recurrent laryngeal nerve paralysis afterward which lasted for about six months, with complete recovery and, when seen about a year after the operation was in perfect health. I do not think we can blame the tracheotomy on the removal of the foreign body. I do not believe it is quite fair to imply that tracheotomy was the result of removal of the foreign body.

The smallest infant upon which I have performed a successful tracheotomy weighed 4 pounds 15 ounces. In this case the complaints were of difficulty in nursing. It was impossible to pass the bronchoscope. A laryngeal grasping forceps was inserted and opened, rather forcibly dilating the stenosis, which was due to tough fibrous adhesions of the vocal cords in the anterior three-fourths of the larynx. It was obviously impossible to get a 3.5 mm bronchoscope through, so the smallest intratracheal catheter was slipped through easily into the trachea. These tiny catheters should be a standard set-up in the bronchoscopic operating room, for just such cases, where the bronchoscope cannot be passed. This child's larynx was subsequently

dilated and after two year the tracheotomy tube was removed; that was two years ago. In the newborn the time of greatest work is when nursing, so that while the infant may be relatively asymptomatic, when nursing there is choking, coughing and gagging, and that may be the presenting symptom of laryngeal obstruction.

I know of one fatality which I think was due to frank neglect. The infant was tracheotomized on the second day of life and died on the third day. A special nurse had been assigned to the case. The supervisor removed the special nurse; the child was seen about 20 minutes later and seemed to be all right, and 20 minutes afterwards was found dead. At autopsy the tracheotomy tube was found to be completely full of secretions. Great emphasis must be placed upon constant attention to the newborn with a tracheotomy.

With regard to postoperative care: At St. Luke's Hospital we have found that the newer types of aerosol generators with a tracheotomy mask have simplified the method of supplying humidity to these children, using oxygen or compressed air as a means of producing aerosol and preventing thickening and drying of secretions.

DR. BURTON J. SOBOROFF: One of the serious complications of tracheotomy in young infants and children is mediastinal emphysema and pneumothorax. I should like to ask Dr. Holinger whether this occurred in any of the cases in this series and, if such complications did arise, what therapy was used.

DR. PAUL H. HOLINGER (closing): I should like to thank Dr. Andrews and Dr. Soboroff for their remarks. In reply to Dr. Andrews, certainly there was nothing implied in the discussion of the infant tracheotomized to remove the safety pin. Obviously when a tracheotomy is indicated it should be done. It was so described merely to stress the fact that in foreign body work in small infants, a tracheotomy is the great exception and not a routine procedure. As you know, there are institutions in which a tracheotomy is performed on any child under two years of age who has a foreign body, and the foreign body is removed by lower bronchoscopy rather than through the larynx. Foreign body work is a peroral procedure for the most part, in spite of the fact that the infant may be under one year of age or, as in a number of our cases, less than a week old when a foreign body has been removed from either the air or the food passages.

With regard to nursing care, Dr. Andrews has mentioned the key to the entire problem—the teamwork which is essential between resident, nurse and, from the economic standpoint, the administration. The economics of the management of such children is an ex-

tremely serious problem. The fact that they do require constant care is the key to the problem and for that reason we attempt to teach the mother as early as possible the management, care, aspiration and cleaning of the tracheotomy tube, so that she may take the child home as soon as possible and continue its care at home. She is better able to give the child 24 hour nursing care than is the hospital where, as you all know, from day to day the problem of economics becomes greater. The mother wants to take care of the child and can be taught to do so, even a small tracheotomized infant.

No anesthesia is used for the endoscopic procedure; the bronchoscope is used to establish the airway and for the tracheotomy itself, generally, novocaine infiltration is all that is necessary.

Dr. Soboroff mentioned the problem of mediastinal emphysema and pneumothorax. One case of extensive emphysema occurred in the group and no cases of pneumothorax. I think one of the principal reasons for this is that the airway was established bronchoscopically before beginning the tracheotomy itself. There was occasional subcutaneous emphysema, of course, as part of the tracheotomy procedure, but as a rule this is not a significant complication provided the incision is left open and not sutured tightly around the tracheotomy. With the airway established by the bronchoscope, there is no sucking of air into the mediastinum as would occur with an obstructed airway during exposure of the trachea. I think that occasionally extensive subcutaneous or mediastinal emphysema or a pneumothorax are due to the tracheotomy being performed too low; the head is hyperextended when the bronchoscope is in place and, if the tracheotomy is done too low under those circumstances it allows the incision to slide down to the mediastinum when the bronchoscope is removed and the head brought forward. Mediastinal emphysema then occurs. This serious complication, reported in some articles, can be avoided with a proper tracheotomy in the usual position.

HARRIS PEYTON MOSHER, M.D., D.Sc., LL.D.

1867-1954

Dr. Harris Peyton Mosher died in the Salem Hospital, near his Marblehead home in Massachusetts, November 4, 1954. He had been in failing health and that morning had undergone surgery, from which he did not rally. Services were held the following Sunday afternoon in the Harvard Memorial Chapel in Cambridge. Among the large number of physicians present to pay their last respects were many of his former associates and students. As this group gathered for a few moments on the chapel steps afterwards, the oft-heard remark was "This marks the end of an era in otolaryngology."

Dr. Mosher was born October 21, 1867 in Woodfords, Maine, the son of Andrew and Julia Harris (Woodford) Mosher. His boyhood was spent in Woodfords. After graduating from Deering High School, his father, who was a civil engineer and an inventor, responsible for much of the development of the great United Shoe Machinery Corporation, moved his family to Boston. There the young man entered the Boston Latin School. After graduating he then spent a year in the Massachusetts Normal Arts School. This was followed by four years in Harvard University and four years at Harvard Medical School. He received his M.D. in 1896. He served a surgical interneship at the Massachusetts General Hospital and later as Interne at the Boston Lying-In Hospital. The Spanish-American War gave him his first experience in military medicine when he served as contract surgeon at Montauk Point.

After four years of general practice in Boston he decided upon otolaryngology as the field most suited to his talents. He had become interested in the specialty from serving in the Nose and Throat Out-Patient Department at the Massachusetts General Hospital. He later went to Germany, taking post-graduate courses in Berlin and Halle.

Returning home he renewed his ties with the Massachusetts General Hospital as Laryngologist and became associated with the Massachusetts Eye and Ear Infirmary; an association which he continued, in spirit, even after retirement from active practice, until the day of his death. In 1922 he became Chief of Otolaryngology at the Institution, retiring in 1939. From his first year of practice when he became Assistant in Anatomy in the Harvard Medical School, he was continuously associated with the school until he retired. The last

MARCH, 1932

PHOTO DR. WERNER MUELLER

fourteen years he was Professor of Otolaryngology: the first professorship of otology and laryngology as one department at Harvard.

The combined position of Professor at the school and Chief of Otolaryngology at the Infirmary gave full play to Dr. Mosher's talents and raised both the hospital and the department at the school to heights never before attained. His residency program became the goal of every budding specialist, especially the sons of men prominent in Otolaryngology. His post-graduate course at the school, which he conducted for 35 years consecutively, except for the period of the First World War, was famous throughout the medical world.

In 1915 Dr. Mosher joined the first Harvard Unit and served in the British Army in France at General Hospital No. 22. With this country's entry into the war he was commissioned as Major and assigned to the Surgeon General's Office, where shortly he became Chief of Otolaryngology with the rank of Colonel. As such he was responsible for the organization and conduct of departments of Otolaryngology in the Army hospitals. This was a revealing experience for him and one which had tremendous effects upon the specialty. Certain inadequacies in training and qualifications, in many cases, were all too apparent. The resulting development of modern otolaryngology, with high standards of teaching, largely may be attributed to his dynamic leadership One outstanding example is the American Board of Otolaryngology of which Dr. Mosher was the first, and 22 years, the only president.

Dr. Mosher was president of both the American Laryngological Association and the American Laryngological, Rhinological and Otological Society in 1920; of the American Broncho-Esophagological Society in 1921; of the American Academy of Ophthalmology and Otolaryngology in 1929; of the American Otological Society in 1938. In addition he served as Chairman of the Section of Otolaryngology of the American medical Association. He also was a Fellow of the American College of Surgeons, and a Corresponding Fellow of the Royal Society of Medicine of London, the British Laryngological Society and the Medical Societies of Paris and of Vienna. He was the guest of honor of each of our four national societies and was the first American to deliver the Semon lecture before the Laryngological Section of the Royal Society of Medicine, receiving on that occasion the Semon Medal from the University of London.

In 1934 he received the deRoaldes gold medal award and in 1947 the Newcomb Award of the American Laryngological Association. In 1937 he was presented with the Gold Medal of the American Academy of Ophthalmology and Otolaryngology.

He was the recipient of honorary degrees of D.Sc. from the University of Pennsylvania, Colby College, and Jefferson Medical School, and of LL.D. from Wayne University.

Dr. Mosher was endowed with the inquiring mind of the true scientist and was indefatiguable in his pursuit of knowledge. He was constantly engaged in research throughout his entire medical career. His work on the esophagus is especially outstanding. He published 89 scientific papers. This number in itself may not be particularly unusual but what is unique, is that every single publications is of the highest quality. Perusal of these papers, dating from 1902 to 1951, furnishes a study of the scientific mind through the years, the changing focus, and the broadening scope of vision. Early papers dealth largely with anatomical subjects and with new instruments. Later we find attention devoted more to surgical procedures and techniques. This is followed by papers concerned with the pathology of the specialty. And in the later years we find a definite philosophical trend in his writings, undoubtedly a reflection of the writer's habit of reading "regularly something worth while in non-medical literature."

No one who ever had the privilege of knowing Dr. Mosher could ever forget him. While he sometimes presented a bluff exterior to those who did not understand him, underneath he was the soul of kindliness, generosity, and loyalty. Nobody will ever know of all the many young people he has helped in making possible a medical career. With it all he was frank and outspoken, and always a seeker for the truth. He never could stand for dishonesty or hypocrisy, and his aversion to either was always quite evident. He was unique in his objectivity and his ability to get at the solution of a problem. While he had pride in those things he accomplished and demanded recognition of his priority, he never sought to keep them to himself. Unselfish to the core, he insisted that each of his associates be able to do everything he did. He never held anyone back. And with it all he was a humorous person, who loved a good joke. But heaven help one who told an off-color story in his presence. While known for his wisecracks, as he called them, it may be surprising to the present generation to learn that in his younger days he was the outstanding producer of gags and comedy skits at the banquets of the various societies.

Dr. Mosher was married March 22, 1913 to Mrs. Helen Augusta Rothwell who survives him. Never were two people more devoted to each other and their married life can only be described as idyllic.

His clubs were the Boston Art Club and the Harvard Club of Boston. While not a member of any denomination, he attended both the Congregational and Episcopal Churches.

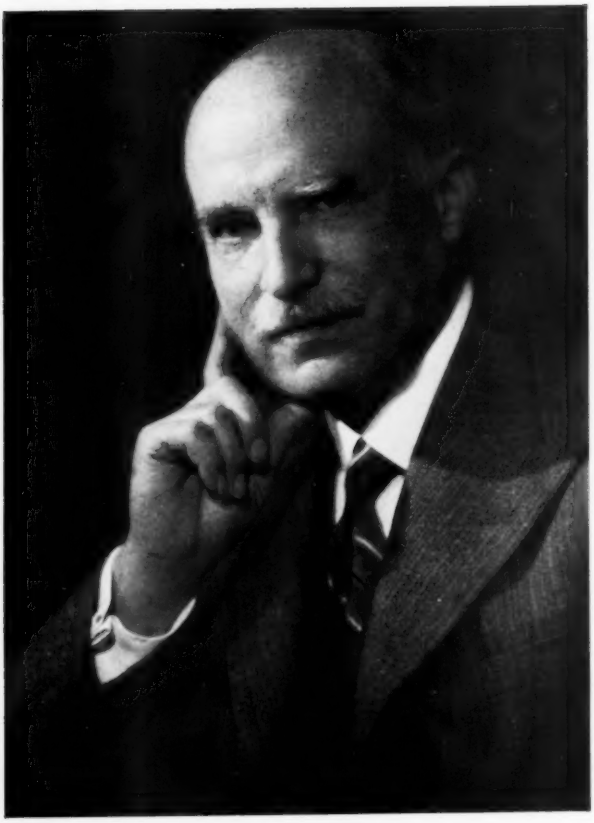
His early training in art enabled him to incorporate in his teaching certain unique features of inestimable value to his students, whereby they made drawings and models from original anatomical and surgical specimens. It also provided him with hobbies with which he occupied his spare moments. He worked in all media.

He once stated that his greatest professional satisfaction was his vicarious fatherhood of some five hundred men in his post-graduate courses in the hospital. It is doubtful if any of us can begin to measure the extent of his influence in otolaryngology. More than any other he has been the leader, the teacher, the crusader.

F. T. HILL.

MEMORIAL NUMBER

The March Number of the ANNALS will be a special issue dedicated to the memory of Doctor Mosher and will consist largely of scientific and biographical articles written for it by some of his friends and former students.



GUNNAR HOLMGREN

GUNNAR HOLMGREN

1875-1954

Gunnar Holmgren, Emeritus Professor of Otolaryngology in the Karolinska Institute Stockholm, and greatly respected friend of oto-laryngologists throughout the world, died in Stockholm early in September, a few weeks before his 79th birthday.

He came from a medical family with a high tradition of public service, his father Fritzhof Holmgren being Professor of Physiology at Uppsala and his mother, Ann Magaret, the well known Swedish feminist.

His appointment to the chair of Otolaryngology in the Karolinska Institute, Stockholm, in 1912 was the first of its kind in Sweden, and when he retired from this post in 1940 he had become one of the world's leading figures in this field of medicine.

His energy and great organizing ability inevitably led to his being called upon to assume heavy responsibilities outside his own particular branch of medicine and from 1933 to 1939 he was Rector of the Karolinska Institute and later was President of the Royal Academy of Science Sweden.

During the time that Gunnar Holmgren was Rector, the new Medical Institute and Hospital were designed and built; and the present magnificent buildings at the Karolinska are a lasting tribute to his work as Rector.

He was also foremost in the field of literature being the founder in 1918 and Editor-in-chief until his death of Acta-Otolaryngologica. He also largely founded in 1929 and guided to success the general medical journal 'Nordisk Medicin.'

In later years he took more and more interest in otology and allied fields of interest and in 1948 founded and became President of the International Conference on Audiology, and many will remember with pleasure the most successful Course in Audiology which he organized and over which he presided with such distinction in Stockholm in 1950.

However, to most oto-laryngologists throughout the world he will be particularly remembered for the part he played in the development of the surgical treatment of otosclerosis, and it will be of interest to recall how this came about.

In 1917, thanks to Gunnar Holmgren's efforts, Robert Barany the Hungarian otologist and Nobel prize winner for Medicine in 1914, was released from the prisoner of war camp in Russian Turkestan where he had been since his capture by the Russians in battle when he was a doctor in the Austrian Army. This release, arranged through the International Red Cross was in exchange for the Russian ballet dancer Nijinsky who was held in Hungary and who was allowed to go to America. Barany came to Sweden where Holmgren found him an appointment in Uppsala which eventually led to the first chair in oto-laryngology in that University.

Barany stimulated Holmgren's interest in the possibility of restoring hearing in otosclerosis by making an opening in the vestibular labyrinth. With typical foresight and fixity of purpose Holmgren concentrated on this work and despite early failures persevered until after about 20 years he was able to see the fenestration operation become an established and successful procedure thanks to the further development of the surgical technique by his pupil Sourdille and the later modification and simplification by Lempert.

Gunnar Holmgren was a great otologist and a great administrator whose memorial lives on in all that he did for Swedish and Scandinavian otolaryngology and medicine and for otolaryngology throughout the world.

He received many Swedish and Scandinavian decorations and was a member of many medical societies throughout the world, including Honorary membership of the American Otolaryngological Society.

He will be remembered with respect and great affection by his many friends throughout the world whose sympathy is extended to his wife Frau Emmy Holmgren and his two sons.

T. E. CAWTHORNE.

Abstracts of Current Articles

EAR

The Effect of Oxygen Lack and Decreased Blood Pressure on the Microphonic Response of the Cochlea.

Gissilsson, L.: Acta Oto-laryng. 44:2:101-118 (Mar.-Apr.) 1954.

Evidence points to the cochlear potentials arising in the hair cells of the organ of Corti. The author's experiments show that the amplitude of these potentials is decreased by lack of oxygen, the decrease varying with the degree of anoxia. This is accompanied by a transient drop in blood pressure but after one to two minutes there is an increase of blood pressure with a further decrease in amplitude of cochlear potentials. Subsequent administration of oxygen increases both blood pressure and cochlear potentials. This is followed by a gradual drop in blood pressure and in amplitude of cochlear potentials. This suggests that the potentials have two factors, one sensitive to lack of oxygen and the other to changes in blood pressure.

Thirty seconds deprivation of oxygen produces reversible injury to the cochlea which becomes irreversible at two minutes. The author feels that many cases of nerve deafness may be explained on the basis of anoxia of the cochlea.

From his experiments he assumes that the cochlear potentials are the result of biological rather than electrical effect.

HILL.

Intratympanic Muscle Reflexes in Psychogenic Deafness.

Jepson, O.: Acta Oto-laryng. Suppl. 109:61-69.

The impedance determination technique of Metz is employed to study the reflex contraction of the intratympanic muscles. In a control group of normal subjects positive responses were elicited, generally at 70-90 decibels. In two cases, later proven to be psychogenic, there was a normal impedance determination. In a third case of hearing impairment following streptomycin therapy but with a suspected functional overlay; this was proven by the impedance test and later confirmed by follow-up audiograms. A fourth case, considered at first to be psychogenic, showed absence of intratympanic reflexes and later proved to be one of genuine hearing loss due to trauma.

HILL.

Influence of the Patulous Eustachian Tube on the Acoustic Impedance of the Ear

Metz, O.: Acta Oto-laryng. Suppl. 109:105-112.

Impedance determination was measured in seven cases of patulous eustachian tubes and proved to be of value in establishing the diagnosis, the intensity of the tone swinging synchronously with respiration.

HILL.

Fenestration and Experimental Nystagmus.

Pursiainen, K. E.: Acta Oto-laryng., Suppl. 112.

A study on 194 patients was carried out to determine: 1, whether fenestration causes changes in the results of rotation and caloric tests; 2, can the results of fistula tests be explained; and 3, what is the relation between rotation, caloric and fistula tests. In the postoperative caloric test cold air (ethyl chloride technique) was employed.

The duration of post-rotation nystagmus in the postoperative tests was shorter toward the operated side, leading to the conclusion that fenestration generally alters the response of the horizontal semicircular canal. The average duration of caloric nystagmus was diminished after operation, with greater difference noted between the response from the operated and non-operated ears, indicating that fenestration generally results in a changed response of the vestibular apparatus. All but five cases subjected to the fistula test gave positive results. The direction was homolateral in 112; heterolateral in 18; varied between the two sides in 4; and was vertically downward in 1.

While one might assume the negative response being due to closure of the fenestra the question of operative damage cannot be ignored, particularly as the study does not include data on the postoperative hearing status. The author frankly states that while "in many cases it seems possible to explain the direction of nystagmus on the basis of cupular deviation due to endolymph flow, there are a number of cases in which the factors influencing the direction cannot be satisfactorily explained by this movement."

HILL.

A Contribution to the Evaluation of Occupational Hearing Impairment.

Neuberger, F.: Monotsschr. f. Obrenheilk. 88:185 (July-Sept.) 1954.

The author directs attention to a new application of Fowler's recruitment phenomenon in the evaluation of occupational deafness.

His observations are based on the differentiation of conductive, cochlear, or retrolabyrinthine deafness by testing the absence or presence of recruitment, as described by earlier workers.

Acoustic traumas are injurious to the sensory epithelium of the cochlea and should therefore show recruitment. Electrical traumas cause damage to the retrolabyrinthine nervous stuctures (C.N.VIII), as ganglia and nerve fibers. Absence of recruitment is expected.

An example is presented of each of the two possible lesions, both following accidents which occurred during telephone switchboard work. Both operators, on different occasions, experienced a sensation described as "sudden shock" in the ear covered by the telephone receiver. The shock was accompanied by sharp pain and the onset of lasting deafness.

Immediate examination of Case 1 revealed a small recent traumatic perforation of the tympanic membrane with an audiometric graph showing "nerve deafness" with recruitment (tested by the binaural method) in the affected ear. Three days later infection of the perforated ear became evident and the recruitment phenomenon disappeared.

In Case 2 the tympanic membrane was found to be normal. The audiometric test revealed nerve deafness without recruitment.

The author interprets Case 1 as an acoustic lesion and therefore cochlear damage. The absence of the at first evident recruitment phenomenon in Case 1 after three days is explained by the onset of middle ear infection causing conductive deafness.

Case 2 is interpreted as retrolabyrinthine damage of structures along the pathway of the VIIIth nerve, which is considered typical for electrical insults.

In view of the masking middle ear infection in Case 1 the importance of early examination for the differential diagnosis of electrical or acoustic deafness is stressed.

FISCHER.

Experimental Investigations of the Effect of Cortisone on Surgical Defects of the Labyrinth Capsule in Guinea Pigs.

Hussarek, M. and Novotny, O.: Monatsschr. f. Ohrenheilk. 88:7 (Jan.-Mar.) 1954.

The paper presents a contribution to the problem of keeping the operative labyrinth window open following fenestration surgery.

The authors are convinced that the occasionally encountered postoperative closure of labyrinth windows (clinically found as poor or no hearing improvement) is due to a serous labyrinthitis causing fibrosis and finally ossification.

As experience in recent years has shown that cortisone suppresses inflammation and formation of fibrosis. Although cortisone has failed to halt the progress of otosclerosis, it has proved valuable in suppressing the postoperative serous labyrinthitis following fenestrations, as found by H. Bell.

The experiments in the presented paper are based on these findings as well as on the theory that the serous labyrinthitis is a forerunner of the eventful ossification of the fenestra novum. The authors fenestrated the labyrinths of several guinea pigs. In the postoperative course cortisone therapy was instituted at varying and well-recorded intervals.

Group 1 received cortisone for two weeks beginning one week after the operation. Three weeks post-operatively, the animal was sacrificed and horizontal sections of the labyrinth were prepared and compared with a control animal which had received no cortisone.

Group 2 received cortisone for ten days beginning two weeks post-operatively. Sections were prepared five weeks post-operatively and compared with sections from an untreated animal.

Group 3 received cortisone for two weeks beginning three weeks post-operatively. Here, too, the animal was sacrificed five weeks post-operatively together with the control animal and sections of the labyrinth were prepared.

Group 4 received cortisone immediately after surgery, and sections of the labyrinth were prepared two weeks after the operation.

Groups 5, 6 and 7 were not treated with cortisone until five, six and seven weeks post-operatively, respectively.

Several photomicrographs attached to the paper show the progress of the postoperative fibrosis and finally ossification of the windows in untreated or late treated animals, compared with little or no formation of connective tissue in animals treated immediately following the operation.

Cortisone trials on fenestrated patients have shown encouraging results, the authors report, providing the cortisone therapy was begun immediately after the fenestration operation.

Cortisone therapy instituted after onset of fibrosis or even ossification was unsuccessful.

The suggested dose in the immediate postoperative treatment is 100 mg of cortisone daily for about four weeks.

FISCHER.

Cholesteatoma in Congenital Atresia of the External Auditory Meatus and Microtia.

Wolfram, G.: Monatsschr. f. Ohrenheilkunde 88:170, 1954.

A case of chronic purulent discharge from an ear with an underdeveloped pinna and congenital atresia of the ear canal is described. The deformed ear canal consisted of a very narrow fistulous tract, out of which fetid smelling pus exuded.

Roentgen films of the mastoids show a well pneumatized mastoid on the left. The diseased right side also showed pneumatization to some extent: Two or three large cells could be seen next to a large cholesteatomatous defect.

The radical mastoidectomy revealed a large cholesteatoma in the medial end of the external auditory canal. The tympanum was found as a narrow slit with rudimentary ossicles but no cholesteatoma.

Pus and necrotic epithelium in the medial end of the external auditory meatus had found no way to the outside due to the stenosis of the infected fistulous canal, the author explains. A cholesteatoma had developed and pushed toward the middle ear.

This explanation seemed supported by the x-ray findings of an apparently good pneumatization of the mastoid, into which the cholesteatoma had expanded from the external canal.

FISCHER.

NOSE

Malignant Tumors of the Nasal and Paranasal Cavities.

Lachmann, J.: Oto-Laryngologica Israeliana 3:51, 1953.

The authors report 32 cases of malignant tumors of the nasal and paranasal cavities observed at the Radium Institute and the Ear, Nose and Throat Department of the Hadassah University Hospital, Jerusalem.

A total of 5200 cases of malignant tumors were treated at the Radium Institute since 1934. Of these, malignancies of the nasal cavity and sinuses accounted for 30 cases or 0.58 per cent.

The importance of unilateral nasal obstruction, bleeding, fetid discharge and unilateral neuralgia pain of the head and neck are stressed.

Electrosurgical resection by x-ray and radium radiation are advised for all lesions confined to the bony cavity of origin.

Lesions penetrating into the soft parts of the face are treated by radiation alone, as are lesions such as lymphogranuloma, lymphosarcoma, and sarcomatosis.

A table showing the 13 operated cases lists 7 living from 2.5 to 18 years after surgery; five expired three weeks to two years post surgery, and one was not available for follow-up.

Sooy.

Rhinoplastic Applications to Submucous Resection of the Nasal Septum. Surgical Anatomy: Technique of a Modified Procedure.

Rowland, Alan L.: A. M. A. Arch. Otolaryng. 59:739-743 (June) 1954.

The submucous resection, the most frequently performed operation remaining wholly in the field of the otolaryngologist, should be reviewed in the light of recent studies of the physiologic functions of the nose. Rhinoplasty has seen many recent developments in its function as a means of restoring disturbed nasal mechanism to a normal status. The traditional operation for submucous resection has become so fixed that little has been added to the literature on it in recent years.

The surgical anatomy of the septum must be kept in mind when attempting to correct its deviations and this is reviewed and it is emphasized that in almost every nasal disfigurement, the septal cartilage is involved. Submucous resection, done according to rhinoplastic principles, seems to the author to more successfully correct allied malformations than the traditional operation. This operation disturbs the normal physiologic structure and function of the nasal mucosa as little as possible, which is not true of the traditional type. The only area in which the respiratory epithelium will not be injured and the area most logical anatomically is defined as a line following the caudal end of the cartilaginous septum and immediately adjacent beneath it. This is the transfixion incision used by the rhinoplastic surgeons for repair of malformations of the lower cartilaginous vault.

Exposure of the entire base of the septum is greater with the transfixion incision than the Killian-type. The cartilage is easily visualized and, if dislocated, may be easily resected and replaced in the midline. Access to the bony portion of the septum is simpler. The value of this type of incision in correcting moderate displacement of the lower cartilaginous portion or in deviation involving the tip of the nose is discussed. There is a trauma involved in the procedure because of the greater exposure. Greater ease in surgical performance results in less chance of encrusting perforations.

The author's technique is described in detail.

HILDING.

Treatment of Nasal Polypi, with Comments on Frontal Sinus Surgery.

Harpman, J. A.: A. M. A. Arch. Otolaryng. 59:655-671 (June) 1954.

The author, from Warwickshire, England, points out that the usual types of nasal polypi are those of allergic or infective nature or often the latter superimposed on the former. Under local or general anti-allergic treatment small, early allergic nasal polypi do disappear, but this is unusual. He feels the cure of nasal polypi lies in removal or otherwise treating the diseased tissue from which the polypi arise.

If only a few polypi are present, simple removal may result in cure. He has found recurrence less likely if removal is followed by gentle curettage of the base of the polypi. In many cases, removal of polypi, intranasal ethmoidectomy and sometimes removal of most of the middle turbinate is a satisfactory procedure. The author describes his method of doing the intranasal ethmoidectomy; he finds ethmoidectomy unsatisfactory when there is much suppuration and dangerous in the presence of bony dehiscences in the roof or lateral walls of the ethmoid labyrinth. He mentions that intranasal ethmoidectomy is only very rarely complete and, when purulent ethmoiditis exists, suppuration continues after incomplete ethmoidetomy. Cicatricial obstruction of the frontonasal duct may follow intranasal ethmoidectomy.

Harpman has found where polypi arise from the antra, and other sinuses are normal the Caldwell-Luc operation, with removal of antral mucosa, is generally curative. Deep x-ray therapy to the antra, after a second removal of distressed lining, has arrested the progress of the disease in two patients whose antra refilled twice after a Caldwell-Luc operation.

Transantral ethmoidectomy has been done by the author many times. This approach is only adequate for the lower part of the middle and posterior ethmoid cells. Occasionally he has found intranasal ethmoidectomy and partial removal of diseased antral lining through an intranasal antrostomy successful in curing nasal polyposis.

In a number of patients with chronic polypoid purulent polysinusitis, Harpman found the best treatment often consisted in removal of the diseased lining from all affected sinuses. He feels when a sinus is sufficiently diseased to warrant radical surgery, its lining is so diseased that operation will not be entirely successful in curing the complaints unless the lining is removed. He feels that two combinations of radical operations are most successful in this condition, when simpler measures fail: Ferris Smith, Lynch, Sewall or Luongo and the Caldwell-Luc operation and the Ferris Smith and Norman Patterson types of operations. He presently favors the latter combination which permits more thorough exenteration of the inferior ethmoids and because the same approach can be used for antrum as well as other sinuses. His technique is outlined in detail; chemotherapy, anesthesia, dressing of the wound and postoperative nasal treatment are described. The complications of the radical sinus operation are listed and results noted. The latter includes reduction of intranasal suppuration in all cases and cure in many; in many, sinusitis, pains and headaches were cured; no patient had a recurrence of nasal polypi; there were no intracranial complications or osteomyelitis.

In general Harpman feels that the extent of surgery depends on the severity of the condition. Radical sinus surgery should only be done when simpler methods fail. He concludes that when a frontal sinus is so infected that conservative measures fail to relieve it and external surgery is required, the infected lining should be removed. Drainage of the sinus cavity into the nose after the operation is required only until healing has taken place. The radical sinus operation, described by the author, he feels, fulfills these criteria. He quotes Sebileau who stressed that when all diseased lining had been removed from a frontal sinus, drains are not required. Harpman found when the mucosa had been completely removed from a small frontal sinus the whole cavity of the frontal sinus became completely filled with new fibrous and bony tissue as healing took place.

It is interesting to note that this type of surgery, which was done in the United States about 40 years ago, has been largely discontinued in this country today.

HILDING.

Problems In Vasomotor Rhinitis.

Harkins, Herbert P.: A. M. A. Arch. Otolaryng. 59:531-535 (May) 1954.

The author uses the term vasomotor rhinitis to cover patients having primary symptoms of itching, paroxysmal sneezing, profuse watery nasal discharge, uniform swelling of turbinate bodies, grayish white mucosa and eosinophilia but no positive skin reactions. Change in atmospheric temperature or humidity or eating seem to be the precipitating factors in the largest number of patients. Pale, boggy nasal mucosa, copious watery discharge, hypertrophied inferior turbinates with "mulberry" posterior tips characterize the condition. The presence of eosinophils in the nasal secretions is the most important single diagnostic criterion. Eosinophils in quantity and alone indicate uncomplicated allergy. Neutrophils in large numbers denote infection, and a combination of the two indicates the coexistence of both allergy and infection. The role of the eosinophil in allergy is known but it has been shown to be a carrier of histamine, though it is not known whether histamine is carried to the tissues or away from them by the eosinophil. Intradermal tests with histamine diphosphate produce increased histamine reactions in patients suffering from vasomotor rhinitis.

Harkins advises careful examination before and after shrinkage of the nasal mucosa. If polypi are present, he advises surgical removal and cautions that it is uncommon to find multiple polypi in the nasal passages without similar, and often larger, growths being present in the nasopharynx. A submucous resection is in order if true deviation of the nasal septum or spurs are present blocking the airway. If the inferior turbinates become small after shrinkage, electrocoagulation of the engorged tissue suffices and is the most frequently performed surgical procedure in the treatment of vasomotor rhinitis and is one of the most successful. It gives excellent permanent results. The most gratifying recent advance the author feels is the histamine desensitization and nonspecific antigen therapy. He finds these patients are greatly helped by fortified house-dust injections given subcutaneously or intradermally along with immunizing doses of histamine diphosphate. Optimum dosage method is used after skin titration for the selection of the proper dilution, resulting in more effective therapy lasting over a longer period of time. The dosage schedule is detailed by the author. While awaiting full therapeutic effect from the immune therapy, antihistaminic drugs and bland vasoconstrictive nasal sprays may be used to relieve symptoms, but should be gradually withdrawn as soon as immunologic therapy has become effective.

HILDING.

Reactions in the Nasal Mucosa. Relation of Life Stress to Chronic Rhinitis and "Sinus" Headache.

Wolf, Stewart: A. M. A. *Arch. Otolaryng.* 59:461-475 (Apr.) 1954.

Experimental work has shown that a reaction of defense in the nasal mucosa—hyperemia, engorgement, hypersecretion and obstruction in the nose—is produced by physical trauma such as inhalation of ammonia, and of pollen to which an individual is sensitive and by exposure of experimental subjects to a non-specific threat not directed at the respiratory passages. Investigation was directed toward elucidating the circumstance when a situational threat, not involving physical trauma, was employed and determining whether this would induce this defense pattern in the nasal mucosa. A threat was utilized which depened upon communication and understanding of symbols which, because of past conditioning experience, had acquired special meaning for the individual. It became clear, as a result of this investigation, that situational threats called forth the same responses in nasal mucosa as had the locally applied noxious stimuli. Added to the above described nasal defense reaction were the ever present bacterial and virus organisms which made the complicating factor of infection possible. An asthmatic attack was also induced in one subject while the nasal structures were observed under a situation of stress.

Data concerning the personality of each subject were correlated with the symptoms and with the observations of nasal functions. A high percentage of onsets and recurrences of nasal troubles coincided with episodes of situational stress and conflict and a fairly clear-cut pattern of attitude and behavior was recognized among them. Their reactions in meeting threats were predominantly defensive. It was felt by the author that the "shutting-out" pattern in the nose reflected a general reaction of non-participation, rather than aggression, in dealing with their problems. He concluded that the pattern of reaction involving the upper respiratory tree is a defensive one of shutting out and washing away at the head of the organism; it is effective and appropriate in dealing with an atmosphere of dust, smoke, fumes, etc. but less so with an atmosphere of hostility from human beings. But, however, inappropriate it is, it does occur and the various factors seem to work in concert and may set up a vicious cycle. Wolf thinks that transitory nasal hyperfunction, whatever its etiology, can be tolerated well but sustained engorgement of tissues with obstruction not only leads to pain and discomfort but may predispose to development of polyps and establishment of infection locally in the nose, paranasal sinuses and bronchi.

To study the character of the tissue change, biopsy specimens were taken, under controlled conditions, from the inferior turbinate of a chronic rhinitis sufferer, first on one side during a normal period, then on the opposite side at the height of a frustrating interview. The first section showed an essentially normal structure with moderate round cell infiltration; the second showed mucous glands filled with secretion, vascular and lymphatic channels prominent and dilated and evidence of edema. Cell counts were made of peripheral blood and nasal secretions before, during and after an attack. With stress there was marked eosinophilia locally and in the peripheral blood and, in the subject described, also a purulent response. The author also found increased fragility of the membrane and lowered pain threshold accompanying sustained nasal hyperfunction induced by symbolic threats.

Adverse life situations were compared with pollen as to effects upon the eosinophilic reaction. The data appeared to show that various factors provocative of nasal hyperfunction with symptoms of hay fever exerted an additive effect.

In an effort to clarify the mechanism of these changes, several individuals were subjected to stellate ganglion block, eliminating sympathetic impulses affecting the nasal membranes and allowing cholinergic fibers of the greater superficial petrosal nerve to act uninhibited. It was found that the unopposed parasympathetic supply induced a state of moderate nasal hyperfunction and the partially denervated membrane was even more reactive to noxious stimuli than usual.

Wolf calls attention to the fact that the bronchial tube is lined with the same type of mucous membrane as the nasal structures and the pathology of asthma is similar to that of rhinitis—membrane engorgement with edema, hypersecretion of mucus, and obstruction. Many of the patients had both diseases. It was equally possible with those with asthma to turn off and on attitudes by manipulating the interviews.

The author cautions that he does not mean to imply that all nasal diseases arise from stress situations but the factors involved in this reaction are important and may affect the course of other processes regardless of the precipitating incident.

HILDING.

PHARYNX

Recurrence Rate of Adenoids.

Jorgensen, A. and Zilstorff-Pedersen, Kaj.: Acta Oto-laryng. Suppl. 109:70-73.

While most writers feel that the recurrence of adenoids nearly always is due to incomplete operation and advocate visual control of the procedure by elevation of the soft palate, the outhors consider that complete removal is impossible because "of the gradual transition of adenoids into the normal lymphoid tissue of the mucous membrane." The report is concerned with a follow-up study of 312 children operated upon with the "blind" technique. Only 4.5 per cent were stated as showing recurrence and of these only 2.9 per cent had symptoms of significance. It was felt that the low incidence of recurrence was due to all of the operations being performed by specialists and the "blind" technique gave satisfactory results in expert hands. They stress the importance of consideration of other factors such as sinusitis, otitis media, allergy, tonsillitis, etc., causing symptoms postoperatively.

One could wish that the method of re-examination could have included the use of the electric nasopharyngoscope rather than posterior rhinoscopy and "if required" digital examination. Certainly the reported low recurrence rates, especially with the "blind" technique, hardly is in line with the experience of most writers on the subject.

HILL.

Operative Treatment of Complete Adductor Paralysis. Experience with Musculatomy of the Sterno-thyroid.

Sjostrum, L.s Acta Oto-laryng. Suppl. 109:141-142.

Two cases of unilateral adductor paralysis following thyroidectomy are reported in which section of the sterno-thyroid muscle resulted in apparent improvement. It is difficult to see how section of this muscle could do other than free the position of the thyroid cartilage from the effect of postoperative scarring. Certainly it could have no effect on muscles innervated by the recurrent laryngeal nerve. The reported improvement came later and might be due, at least in part, to the compensatory action of the uninvolved cord.

HILL.

ESOPHAGUS

Spontaneous Rupture of Esophagus.

Large, J. M.: Lancet 529 (Sept.) 1954.

Spontaneous rupture of the esophagus though rare has several significant clinical characteristics.

An illustrative case is reported involving a 54 year old man who was seized with an agonizing epigastric pain while vomiting after ingesting a sandwich and two pints of beer. Clinically he showed shock, dyspnea, cyanosis, and abdominal rigidity. The admitting diagnosis of perforated peptic ulcer was soon changed to coronary occlusion because of the cyanosis, then one and one half hours later to perforated esophagus when emphysema of the neck developed and radiography of the chest showed a left pneumothorax. Left thoracentesis revealed air and fluid which smelled of beer and gastric secretion.

Because of the profound shock only supportive measures and catheter drainage of the left pleural cavity were used.

Death occurred 16 hours after the accident and necropsy showed a 5 cm slit in the left postero-lateral esophageal wall. Both lungs were completely collapsed.

The literature is reviewed and the condition found to be 100 per cent fatal prior to 1947. Since this time eleven patients were found who survived the accident; eight were treated early by thoracotomy and primary suture, three by pleural drainage. In the latter the diagnosis was not made for two and one half days to three weeks after occurrence.

The author concludes that the severe shock and cyanosis once the diagnosis is made, does not preclude immediate thoracotomy and direct suture of the esophagus. The symptoms improve rapidly after opening the chest under controlled respiration anesthesia.

Pleural drainage is of value primarily where the diagnosis is established after 24-36 hours and infectious edema makes primary suture impractical.

All cases treated in the first 24-36 hours only by pleural drainage proved fatal

Sooy.

Gastro-esophageal Incompetence In Children.

Astley, Roy and Carre, Ivo J.: Radiology 62:351-362 (Mar.) 1954.

The authors present findings based on a study of 115 cases of vomiting in infancy and childhood due to weakness of the valve mechanism between the esophagus and stomach. Eighteen radiographs demonstrate that this gastro-esophageal incompetence is due to a "sort esophagus" and a minor degree of partial thoracic stomach. Vomiting is the outstanding symptom and usually dates from the neonatal period and the vomiting may contain altered blood. Several examinations may be necessary to establish the radiologic identification of the stomach above the diaphragm. In this group were 5 children with pyloric stenosis in addition to the partial thoracic stomach. Early diagnosis and prompt institution of conservative treatment should reduce the number who develop esophageal strictures and need radical surgical treatment. Treatment consists of nursing in a propped up position maintained by a special harness and giving thickened feedings if vomiting persists. Twenty-five references are given.

JORSTAD.

MISCELLANEOUS

A Study of the Post-Operative Course After Parotidectomy with Preservation of the Facial Nerve.

Hamberger, C. H. and Laage-Hellman, J. E.: Acta Oto-laryng. Suppl. 109:49-55.

The postoperative complications of 34 parotidectomies and 10 "partial" parotidectomies were of a transitory nature and considered as no contraindication to operation if carefully performed. These complications were temporary facial paralysis, remaining partial paralysis, salivary fistula and an auriculo-temporal syndrome of perspiration, redness and burning pain in the preauricular region; which occurred in one case. Because of the potential malignancy of mixed tumors and the possibility of multiple origin, parotidectomy with preservation of the facial nerve was considered to be the operation of choice.

HILL.

Herpes Zoster Oticus with Concommitant Disturbances in the Cranial Nerves, V, VII, VIII, X and XII.

Kawaoka, A. and Ishikawa, K.: Jour. of Otolaryng. (Tokyo) 26:23 (Feb.) 1954.

Thirty-three cases of herpes zoster oticus have been reported in the Japanese literature. Kawaoka and Ishikawa add another case of

their own which developed simultaneous lesions in the V, VII, VIII, X and XII cranial nerves.

The patient was 55 years old, male. The initial symptom was left hemicrania, double vision, dysphagia and hoarseness. On admission to the hospital, the patient presented a typical left Ramsay Hunt's syndrome. There were facial palsy, hepatic lesions of the tympanum and the external auditory canal, impairment of the low tone perception, complete loss of caloric response on vestibular stimulation on his left ear. There was sagging of the left half of the soft palate, hemiparesis of the tongue and left vocal cord paralysis. There was a marked increase in lymphocytes in his spinal fluid. The patient was placed on aureomycin by mouth and vitamin B_1 intrathecally and showed a marked response.

HARA.

A Topographic Approach to the Roentgenologic and Pathologic Examination of Laryngopharyngeal Tumors.

Fletcher, Gilbert H., Old, Jacob W. and Loquvam, George S.: Radiology 63:3: 361-380 (Sept.) 1954.

As a supplement to the classical methods of clinical examination of the laryngopharynx the authors describe the technique of a topographic roentgen study. They present nine representative cases. In these they made topographic histologic sections to demonstrate the value of the preoperative radiographic impression and more adequate examination of the margins of surgical excision. A lateral soft tissue roentgenogram, atomogram, photograph of a longitudinal section of the specimen, and a line drawing to demonstrate the lesion or defect is included with each of these procedures, with descriptions thereof for each nine cases.

From these findings they arrived at a classification which takes into consideration operative criteria, natural history of the tumor and its predictable course. Such a classification based on the site of origin has considerable bearing on the choice of treatment since predictable clinical behavior can be correlated with the particular site. Proper interpretation of data obtained by palpation, laryngoscopy, lateral soft tissue films and tomograms will permit a diagnosis of tumor extrusion as well as the anatomic site of origin in a high percentage of cases.

All tumors originating in the supraglottic region, epilaryngeal area and piriform sinus are treated routinely by a panlaryngectomy followed by heavy irradiation, with better results than surgery or radiation alone.

JORSTAD.

Diced Cartilage Grafts.

Peer, L. A.: Jour. Internatl. Coll. Surg. 22:283 (Sept.) 1954.

Diced cartilage grafts, as the name implies, were originally obtained by longitudinal slicing and subsequent "dicing" of rib cartilage. In practice, however, thin cartilage shavings are found to pack together more evenly without presenting sharp corners which may produce prominence beneath the overlying skin.

Microscopic examination of diced cartilage grafts after transplantation was made at various intervals up to four years. The sections demonstrated that the spaces between the small cartilage segments were occupied first by blood and later by ingrowing connective tissue accompanied by numerous small blood vessels. This was later supplanted by a thin layer of connective tissue thus preventing contracture of the cartilage mass.

Autogenous diced cartilage grafts showed living chondrocytes, a normal appearing matrix, and occasional formation inside or outside of the cartilage. In general, there was an absence of invasion and absorption. Preserved diced cartilage grafts, however, showed definite invasion and absorption.

Diced cartilage was first applied by the author to frontal bone defects and the technique consisted of elevation of the scalp using a "T" incision with exposure of the defect. Diced cartilage was placed generously within the defect and the overlying scalp was closed over this mass.

The technique of using diced cartilage to form the cartilaginous structure for the congenital absence of the auricle is beautifully illustrated. A perforated tantalum mold in the shape of the cartilaginous framework of the external ear is filled with diced cartilage and the two halves of the mold tightly screwed together. The mold is then embedded in the fatty tissue of the lower abdominal wall and allowed to remain for a period of five months. At the expiration of this time the mold containing the cartilage is removed and the metal halves of the mold separated with removal of the cartilaginous ear framework. This is inserted into a previously delayed pocket beneath the skin of the ear region. The position of the cartilage framework is adjusted so that it lies approximately one inch higher than the normal ear to counteract for subsequent sagging. The site of insertion is drained at the lower angle for 48 hours and a firm dressing applied to mold the overlying skin into the contour of the cartilaginous framework.

Diced cartilage grafts have also been used as a substitute for bone grafts to repair mastoid fistulas, to build up prominence of the malar bone in the correction of receding chin, and to restore contour in any bony deficiency of the face.

Grafts for this procedure are packed in a perforated vitallium mold and will conform to the shape of the mold like wet sand. When the perforated mold is buried beneath the patient's abdominal skin, connective tissue and blood vessels grow through the openings in the mold and nourish the cartilage in situ as well as fastening the segments together in a solid plaque. If allowed to remain in place for five months, the plaque will be bound together by mature connective tissue containing tough cartilaginous fibre so that cartilage can be removed from the mold as the solid structure.

The authors also describe their technique in using diced cartilage grafts to form a new articular head for the femur in the presence of ankylosis of the hip joint, as a cartilaginous mold to reconstruct the anterior chest wall in order to protect the heart following heart surgery, and to repair a large spinal cord defect in a congenital spina bifida.

SOOY.

Recent Advances in Retrolental Fibroplasia.

Henry, M.: California Medicine 81:272 (Oct.) 1954.

According to the author, retrolental fibroplasia is the most common cause of pre-school blindness. A condition closely simulating retrolental fibroplasia has been produced in kittens by keeping them in an environment under a high oxygen level. The author feels that the custom of maintaining premature infants in a high oxygen concentration may well account for the abnormally high incidence of this condition among prematures.

Oxygen when ordered for premature infants should be prescribed as a percentage concentration rather than in liters of flow and the concentration should be frequently checked with an oximeter.

Withdrawal of the infant to atmospheric air should be a slow gradual process.

SOOY.

Maintenance Cortisone in Intractable Asthma.

Irwin, John W., Henneman, Philip H., Wang, David M. K., and Burrage, Walter S.: The Journal of Allergy 25:201-209 (May) 1954.

Certain cases of intractable asthma may be relieved of their symptoms and maintained symptom free under cortisone therapy. In the management of these cases the authors follow the following schedule. They administer initially cortisone to suppress the symptoms of asthma completely beginning with 75 mg every 6 hours for 24 hours and then 50 mg every 6 hours until complete relief is obtained. Once this is attained the daily dose is decreased once every week until such time as the symptoms being to reappear and then the daily dose is established at 12.5 to 25 mg above this level. All of their cases to date have required at least one daily dose of cortisone to remain symptom free. After the maintenance level has been determined it fluctuates very little but on occasions the requirements are increased by upper respiratory infections and periods of stress.

The authors call attention to the fact that with the large daily intake of cortisone two or three times the normal rate of secretion of cortisone-like hormone Cushing's clinical syndrome of spontaneous hyperadrenocorticism, at least in part, may eventually be expected. Osteoporosis, one of the most common serious complications should be kept in mind and the patient should be checked periodically for the early symptoms of this condition. This may be done by urinalysis for calcium excretion and roentgenograms of the spine and ribs which in osteoporosis is early demonstrated by generalized increased radiolucency.

Lesser manifestations of long term cortisone therapy are increases of facial hair in female patients and acne in male patients, weight increase with increased fat in the abdominal, facial, supra-claviclar and lower cervical areas. All of the authors' twenty-three patients developed facial rubor and rounding of facial contours.

VAN ALYEA.

Improved Control of Advanced Oral Cancer with Massive Roentgen Therapy.

White, George, Sieniewicz, James and Christensen, William R.: Radiology 63:37-42 (July) 1954.

One hundred and thirty-seven patients with oral cancer were treated at Pondville Hospital between 1944 and 1951. Prior to 1946 conventional radiation therapy (6000 "r" tumor dosage through external ports) controlled 6 out of 54 patients and only one was alive at the end of three years.

In 1947 massive roentgen therapy was instituted. In the group of 137 patients conventional (dosages of less than 5000 "r" to 6,900 "r") and massive (dosages from 7000 "r" to 12,000 "r") roentgen therapy was given. In each case from 4000 "r" to 6000 "r" was given through an intraoral cone. The remainder of the dose was given through lateral and submental external portals after the intraoral therapy was completed. After one year the primary cancer was controlled in 48 per cent receiving massive therapy. Incidence of radiation sequelae did not rise appreciably until the tumor dose exceeded the 10,000 "r" level. Optimal dose appears to be in the range of 9000 "r" to 10,000 "r" delivered over a period of four weeks. Ten tables of statistics are included.

JORSTAD.

The Posture Test.

Nylen, C. O.: Acta Oto-laryng., Suppl. 104, pp. 125-130.

Two techniques are presented, one test easily conducted in the office and the other more complicated, requiring a specially constructed table, and with means of recording, by filming, the ocular movements.

The author stresses the value of testing for positional nystagmus, citing cases of encephalitis and of cerebral tumor where this was the only neurological sign. Direction changing and irregular positional nystagmus indicates a central lesion while direction fixed nystagmus is more suggestive of a peripheral one.

HILL.

Books Received

A Stereoscopic Atlas of Human Anatomy. Section II, Head and Neck.

By David L. Bassett, M.D., Associate Professor of Anatomy, Stanford University, California. In Five Volumes, 8 vo (loose-leaf) with 350 pairs of stereoscopic color transparencies on 50 discs, by William B. Gruber, Sawyer's Inc., Portland, Oregon, 1954.

We have just spent an evening of sheer delight (not to mention several small and intermediate hours of the morning) peering through an electrically lighted viewer at a new collection of color-stereos of head and neck anatomy. Whether the palm goes to the dissector, the photographer, the producers, or even to those remote inventors of Kodachrome is academic; the grand result will be of inestimable value to students, surgeons, examiners and anyone else having to do with the complex structures of these regions.

To begin at the end, there are inside the back cover of each volume ten pockets, each containing a 3½-inch disc, (a standard View-master "reel") with seven pairs of color transparencies. These comprise every type of illustration usually encountered in the anatomical textbooks and in many cases a diversity of views not often to be had in one work. There are isolated bones in enough positions to give one the feeling of turning them about in his hand; and there is a roentgenogram of each. Besides the usual views of this and that, there are series of progressive dissections: for example, of the orbit from an anterior approach, from a superior approach and from a lateral approach; and the same for the ear. There are progressive dissections of the neck from various directions, and some of the whole head and neck from the back.

The dissections are tops in all respects. Arteries are red and veins are blue, as might be expected, but muscles look like muscles and have the color of muscles, and the bones are clean and white. The central nervous system is not represented. It has a separate section devoted to it, which was published in 1953.

The photography deserves special mention. What could have been a routine lighting problem has been handled with discrimination and the angles are chosen to show exactly what is intended. In some of the views the stereoscopic effect appears to have been purposely exaggerated for contrast, with the result that hollow spaces seem

deeper than they are, but this does not detract, and probably accentuates small structures.

The printed sections of the books consist entirely of line drawings of the photographs and on the facing pages anatomical terms, and brief explanatory notes. The drawings are, on the whole, excellent. Some of them, to our way of thinking, could be improved by omitting certain conventional cross-hatchings and other small penstrokes which are confusing because they are not related to the texture of the surfaces represented, and sometimes result in reversing the light contrasts of the photographs.

The discs are capable of projection, either flat or stereoscopic, a distinct advantage over the usual textbook, in that it permits the student to peruse at his leisure the same specimens which were shown in his lectures. No picture, regardless of its clarity (and partly *because* of it) can supplant personal dissection in the training of the future surgeon, but pictures such as these can set him a fine example and in the present dearth of anatomical material can fill a variety of needs.

Distributors: Stacey's, San Francisco; Williams and Wilkins, Baltimore; the price, according to the brochure, "11c a view for 350 . . . dissections."

Atlas of Exfoliative Cytology.

By George N. Papanicolaou, M.D., Ph.D., Clinical Professor of Anatomy Emeritus, Cornell University Medical College. Looseleaf, illustrated. Harvard University Press, Cambridge, Mass. (Price $18.00)

This loose-leaf atlas, prepared by Dr. Papanicolaou himself, is, of course, the last word in authoritativeness regarding the special methods of obtaining and staining cells for the diagnosis of cancer, originated by him and bearing his name. The first chapters are devoted to this technique, and to the general criteria of malignancy as revealed in exfoliated cells. The remainder of the text deals with the special cytology of the female genital system, urinary and male genital systems, the respiratory system, the digestive system, the breast, and certain of the exudates.

Of greatest importance to the diagnostician is the section of large-scale color illustrations of cells in every phase and stage of normality and disease with concise descriptive notes accompanying

each plate. (There are thirty-six of these - 8 x 11). Since the validity of these tests is so greatly dependent upon the experience and judgment of the observer in interpreting what he sees, this manual fills an urgent need.

Peripheral Circulation in Man (Symposium).

Edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch. and Jessie S. Freeman, M.B., B.S., D.P.H., assisted by Joan Etherington, for the Cuba Foundation. Small 8 vo., cloth, xii + 219 pp., 72 illustrations, Little, Brown and Company, Boston, 1954.

This volume on the general physiology of the peripheral circulation contains much to interest the rhinologist, dealing as he does with one of the most sensitive and reactive, and at the same time one of the most accessible of the vascular beds. The book contains eighteen papers constituting a symposium held May 11-13, 1953 (the place is not given) and a liberal discussion.

Audiology: Lectures of the First German Course for Audiologists, Friedberg, October, 1953.

Issued by Prof. Dr. F. Zollner, Director of the University Clinic for Throat, Nose and Ear Diseases, Frieberg, with 14 collaborators. 8 vo., 216 pp., 163 illustrations, George Thieme, Stuttgart, 1954. (In German)

This is a collection of seventeen presentations constituting a course in audiology, the first to be held in Germany. The subject is approached from all angles, including physiological considerations, audiometric apparatus, the special requirements in the various types of deafness, and finally the relation of audiological studies to social adaptation and disability ratings.

Diagnostic Roentgenology of the Skull (Röntgendiognostik des Schadels).

By Dr. Willy Loepp, late Professor of Roentgenology in the University of Konigsberg and Dr. Reinhold Lorenz, Professor in the University of Hamburg, Chief of the Röngenological Institute of the Krankenhaus, Hamburg-Barmbeck. Large 8 vo., cloth, pp. 579, with 696 illustrations, George Thieme, 1954. (In German) (Price DM 97)

A magnificent exposition of the current routine methods and positions in use in the radiography of the skull. Diagrams are plentiful and clear and the roentgenograms are of first quality, although the latter are positives to which the American student will be less accustomed than to the negative prints, duplicating the films, which are customary in this country.

Papers Dedicated to Torsten Skoog on His 60th Anniversary.

Supplement 115 of the Acta Oto-Laryngologica, paper, 303 pp., illustrated, 57 authors participated in this tribute to Dr. Skoog.

Disorders of Voice and Speech (Trastornos de le Voz y de la Palabra).

By J. Perelló, M.D., Vice-president of the International Society of Phonetics and Logopedics, Chief of the Department of Phonetics of the Faculty of Medicine of Barcelona. 8 vo., paper, pp. 132, illustrated, Manuel Marin and Company, Barcelona, Spain, 1954.

The Minute Structure of the Cupola (La Ultrastruttura della Cupola).

By Mario Caossolo, M.D. Volume 65, Supplement XVII of the Archivo Italiano di Otologia, Rinologia e Laringologia. (In Italian. Summaries in English, French and German), 8 vo., paper, illustrated, pp. 71, 1954.

Serological Examinations of Nasal Polyp Fluid.

By Peter Berdal. Supplement 115 to the Acta Oto-Laryngologica. paper, 164 pp., 18 illustrations, 28 tables, 36 case reports.

The Deaf and Their Problems. A Study in Special Education.

By Kenneth W. Hodgson, M.A. (Cantab), with a preface by Sir Richard Paget, Bart., Fellow of the Physical Society. xx + 364 pp. Octavo, cloth, Watts and Company, London, E. C. 4.

Notices

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

The International Congresses of Otolaryngology which had their beginning in Copenhagen in 1928 and which have been held every four years since (with the exception of the war years) are open to every otolaryngologist of good standing in the world, and the following is printed for the information of those concerned.

At the time of this writing each congress is a separate entity. Every participating country may send two official delegates elected by its national society of otolaryngology. These delegates constitute the international committee which considers and acts upon invitations from various countries for the following congresses, and transacts such other official business as may lie without the province of the officers of any specific congress.

The International Committee at its meeting in Amsterdam, June 10, 1953 appointed an Interim Committee to function between the Fifth and Sixth Congresses. This Committee consists of the President and Secretary of the Fifth Congress, Professor Eelco Huizinga and Mr. W. H. Struben, and the President and Secretary of the Sixth Congress, Dr. Arthur W. Proetz and Dr. Paul H. Holinger. In January 1954 representatives of the five national societies of otolaryngology of the United States elected Dr. Proetz and Dr. Holinger as President and General Secretary respectively of the Sixth Congress, with instructions to organize an Executive Committee.

In addition to the above named the executive group now consists of Dr. Frederick T. Hill, Treasurer and Doctors Victor R. Alfaro, Lawrence R. Boies, Louis H. Clerf, Dean M. Lierle, John R. Lindsay, Bernard J. McMahon, Lewis F. Morrison, C. Stewart Nash, LeRoy A. Schall, Harry P. Schenck, and George E. Shambaugh, Jr. The detailed arrangements will be in the hands of this Committee whose separate functions will be announced at a later date.

The Congress will take place in Washington, D. C., May 5-10, 1957. It will open with an inaugural ceremony, Sunday afternoon, May 5. The subsequent Scientific Program will comprise three combined sessions and five or six general sessions, divided into as many sections as may be found necessary.

The official languages of these Congresses have thus far been English, French and German, but for this Congress alone Spanish will be added as a courtesy to the Latin-American members who are expected to attend in large numbers, it being the first International Congress to take place in the Americas. There will be a continuous showing of motion pictures; scientific and commercial exhibits will be open throughout the five days of the meeting.

Banquets, receptions and other entertainment will be on a scale commensurate with the importance of this gathering and there will be auxiliary trips to medical centers of interest in and out of Washington.

Every attention will be given to the special requirements of members from overseas. Sometime in the Fall of 1955 invitations to participate will be mailed to the otolaryngologists of every country in the world.

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct the following examinations:

March 6-10, 1955, in Richmond, Virginia, at the Hotel Marshall.

UNIVERSITY OF FLORIDA

The ninth annual University of Florida Midwinter Seminar in Ophthalmology and Otolaryngology will be held at the Sans Souci Hotel in Miami Beach the week of January 17, 1954. The lectures on Ophthalmology will be presented on January 17, 18 and 19 and those on Otolaryngology on January 20, 21 and 22. A midweek feature will be the Midwinter Convention of the Florida Society of Ophthalmology and Otolaryngology on Wednesday afternoon, January 19, to which all registrants are invited. The registrants and their wives may also attend the informal banquet at 8 p. m.

The Seminar lecturers on Ophthalmology this year are: Dr. William F. Hughes, Jr., Chicago; Dr. Phillips Thygeson, San Jose; Dr. James Allen, New Orleans; Dr. Walter H. Fink, Minneapolis;

and Dr. Milton L. Berliner, New York. Those lecturing on Otolaryngology are: Dr. Paul Holinger, Chicago; Dr. Lawrence R. Boies, Minneapolis; Dr. Edmund P. Fowler, Jr., New York; Dr. Arthur W. Proetz, St. Louis; and Dr. David D. DeWeese, Portland, Oregon.

UNIVERSITY OF TORONTO

The Faculty of Medicine of the University of Toronto offers a combined refresher course in Eye and Ear, Nose and Throat Surgery from April 11 to 15, 1955. The instruction will consist of didactic lectures and surgical demonstrations emphasizing the office practice of ophthalmology and otolaryngology.

The guest surgeons will be: Alson E. Braley, M.D., Professor of Ophthalmology, University of Iowa, Iowa City; Frank B. Walsh, M.D., Associate Professor of Ophthalmology, Johns Hopkins Hospital, Baltimore; Edmund P. Fowler, Jr., M.D., Professor of Otolaryngology, Columbia University College of Physicians and Surgeons, New York; Dean M. Lierle, M.D., Professor of Otolaryngology and Maxillofacial Surgery, University of Iowa, Iowa City.

The staff of the Departments of Ophthalmology and Otolaryngology in the University will contribute to the course. The course will be held for a minimum of 10 and a maximum of 30 students. The fee will be $100.00 payable to the Chief Accountant, Simcoe Hall, University of Toronto. Applications should be made to the Dean of the Faculty of Medicine, not later than February 15, 1955.

UNIVERSITY OF INDIANA

The Department of Otolaryngology, Indiana University School of Medicine offers its annual Anatomical and Clinical Course in Otolaryngology, February 7-19, 1955.

Applicants should address The Post-graduate Office, Indiana University Medical Center, Indianapolis 7, Indiana.

AMERICAN LARYNGOLOGICAL ASSOCIATION

CASSELBERRY PRIZE

A sufficient sum having accrued from the Casselberry Fund for encouraging advancement in the art and science of Laryngology and Rhinology, this sum is now available, in part or as a whole, for a prize award. Theses must be in the hands of the Secretary, Dr. Harry P. Schenck, 326 South 19th Street, Philadelphia, Pa., before March 1, 1955.

Copies of the Transactions of the American Laryngological Association are available for general distribution at $8.00 a copy. Please send request with check to:

Dr. Bernard J. McMahon,
Editor Transactions
8230 Forsyth Blvd.
Clayton 24, Mo.

UNIVERSITY OF PENNSYLVANIA

Announcement is made of the personal fundamental course in Bronchology, Esophagology, Gastroscopy and Laryngeal Surgery at the Graduate School of Medicine, University of Pennsylvania. The course begins September eighth and continues through September nineteenth.

Courses are scheduled usually in January, June and September avoiding conflict with the meeting dates of the National Medical Societies.

Application may be made to Dr. Aims C. McGuinness, Dean, or to Dr. Gabriel Tucker, Chairman of the Department of Bronchology, Esophagology, and Laryngeal Surgery.

TULANE UNIVERSITY OF LOUISIANA
SCHOOL OF MEDICINE

The three year residency in otolaryngology offered at Charity Hospital of Louisiana at New Orleans on the Tulane University of Louisiana School of Medicine service is designed to qualify the holder

for the examinations of the American Board of Otolaryngology and the practice of all phases of otolaryngology and endoscopy.

Candidates must be graduates of a class A medical school and must have completed a minimum of one year of general internship. An additional year of residency in internal medicine or general surgery is desirable but not essential.

All work is under the direct supervision of members of the Tulane Department of Otolaryngology, who are also members of the Charity Hospital Otolaryngological Staff; they are available at all times for instruction and guidance. Basic sciences are offered throughout each year of the residency during the academic year. The resident also participates in the program of the Speech and Hearing Center at the Tulane University School of Medicine.

The hospital year extends from July 1 of one year to June 30 of the following year.

Applications should be addressed to the Chairman of the Department of Otolaryngology, Tulane University of Louisiana School of Medicine, 1430 Tulane Ave., New Orleans 12, Louisiana.

FOURTEENTH JAPAN MEDICAL CONGRESS

The Fourteenth Japan Medical Congress will be held in Kyoto, April 1-5, 1955. Particulars may be had of the Secretary General, Mitsuharu Goto, University Hospital Kyoto University, Kyoto, Japan.

PORTUGESE OTORHINOLARYNGOLOGICAL SOCIETY

On March 20 the Sociedade Portuguesa de Otorrinolaringologia e de Bronco-esofogologia was founded, and held its first meeting in Lisbon under the presidency of Dr. Alberto Luís de Mendonça. The Secretary is Dr. António da Costa Quinta, Avenida de Liberdade 65, 1° Lisbon.

IV INTERNATIONAL CONGRESS

There are a few copies of the Proceedings of the 4th International Congress of Otolaryngology still available for purchase.

The publication contains all of the numerous papers, and is well produced. Copies may be had, at Five Guineas, from

The Secretary
British Medical Journal
B. M. A. House
Tavistock Square
London, W. C. 1.

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American Board of Otolaryngology

President: Dr. Frederick T. Hill, Professional Bldg., Waterville, Maine.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.
Next examination: Richmond, Va., March 6-10, 1955.

American Broncho-Esophagological Association

President: Dr. Clyde A. Heatly, 11 N. Goodman St., Rochester, N. Y.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3, Pa.
Meeting: Hollywood, Florida, March 15 and 16, 1955.

American Laryngological Association

President: Dr. Henry B. Orton, 224 Delavan Ave., Newark, N. J.
Secretary: Dr. Harry P. Schenck, 326 South 19th St., Philadelphia, Pa.
Meeting: Hollywood, Fla., March 13-14, 1955.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Kenneth M. Day, 121 University Place, Pittsburgh, Pa.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Meeting: Hollywood, Fla., March 15, 16, 17, 1955.

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Francis W. Davison, Danville, Pa.
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American Otological Society

President: Dr. D. E. Staunton Wishart, 170 St. George St., Toronto 5, Can.
Secretary-Treasurer: Dr. John R. Lindsay, 950 East 59th St., Chicago 37, Ill.
Meeting: Hollywood Beach Hotel, Hollywood, Florida, afternoon of March 17, forenoon and afternoon of March 18, and forenoon of March 19, 1955.

The American Society of Ophthalmologic and Otolaryngologic Allergy

President: F. Lambert McGannon, Lakewood, O.
Secretary-Treasurer: Michael H. Barone, M.D., Buffalo, N. Y.
Meeting: Palmer House, Chicago, Ill., October, 1955.

VI International Congress of Otolaryngology

President: Arthur W. Proetz, M.D., St. Louis, U. S. A.
Secretary-General: Paul H. Holinger, M.D., 700 North Michigan Ave., Chicago 11, Ill., U. S. A.
Meeting: Washington, D. C., May 5-10, 1957.

International Bronchoesophagological Society

President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U.S.A.

Pacific Coast Oto-Ophthalmological Society

President: Leland G. Hunnicutt, M.D., 98 N. Madison Ave., Pasadena 1, Calif.
Secretary-Treasurer: John F. Tolan, M.D., 3419 47th Ave., N.E., Seattle 5, Wash.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Justo M. Alonso, Montevideo.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U.S.A.

Canadian Otolaryngological Society

President: Dr. G. Edward Tremble, Montreal, Can.
Secretary: Dr. G. Arnold Henry, 170 St. George St., Toronto, Ontario, Can.
Meeting: Lake of Bays, Muscoka, Ont., June 16-18, 1955.

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RESOLUTIO

100 MILL

INSTRUCTIONS Resolution is expressed in terms
film under specified conditions. Numerals in chart ind
"T-shaped" groupings.

In microfilming, it is necessary to determine the red
chart by this value to find the number of lines recorded
ratio, the line above is 100 millimeters in length. Measur
into 100 gives the reduction ratio. Example: the line

Examine "T-shaped" line groupings in the film with
lines recorded sharply and distinctly. Multiply this numb
in lines per millimeter. Example: 7.9 group of lines is
not distinctly separated. Reduction ratio is 5, and 7.9
rily. 10.0 x 5 = 50 lines per millimeter which are not
tions, maximum resolution is between 39.5 and 50 lines

Resolution, as measured on the film, is a test of the
processing, and other factors. These rarely utilize max
exposure, lack of critical focus, and exposures yielding v

ION CHART

OO MILLIMETERS

ssed in terms of the lines per millimeter recorded by a particular in chart indicate the number of lines per millimeter in adjacent

mine the reduction ratio and multiply the number of lines in the nes recorded by the film. As an aid in determining the reduction gth. Measuring this line in the film image and dividing the length le: the line is 20 mm. long in the film image, and $100/20 = 5$.

the film with microscope, and note the number adjacent to finest ply this number by the reduction factor to obtain resolving power p of lines is clearly recorded while lines in the 10.0 group are $\times 5$, and $7.9 \times 5 = 39.5$ lines per millimeter recorded satisfactoich are not recorded satisfactorily. Under the particular condiand 50 lines per millimeter.

test of the entire photographic system, including lens, exposure, utilize maximum resolution of the film. Vibrations during es yielding very dense negatives are to be avoided.